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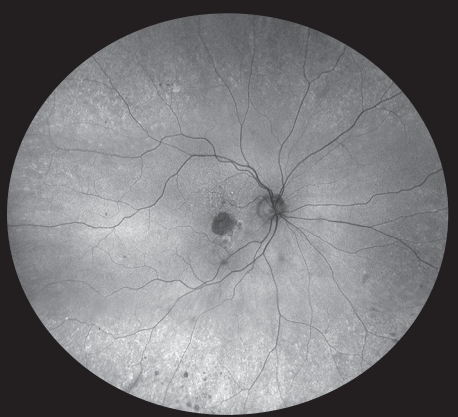

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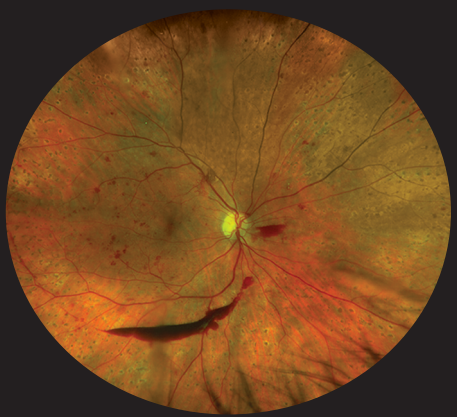

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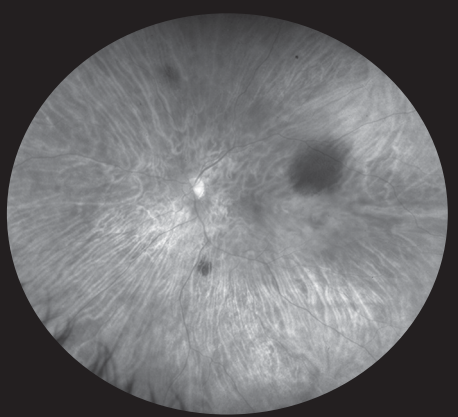

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B. Ralph Chou, MSc, OD, FAAO
Editor-in-Chief/ Rédacteur en chef

Our lead article in this issue is an Executive Summary of the Canadian Association of Optometrists' Clinical Guideline on Optometric Low Vision Rehabilitation. As Canada's population ages, the number of individuals affected by conditions resulting in low vision is expected to increase substantially and with longer life expectancy, their needs for low vision rehabilitation services will be for a much longer duration than for previous generations. This presents a great challenge not only for individual clinicians, but also for those planning for logistics and financing of seniors' health care. The full 80-page Clinical Practice Guideline can be found on the *Canadian Journal of Optometry* (CJO) section of the University of Waterloo's Open Journal System at https://openjournals.uwaterloo.ca/index.php/cjo.

This is the second Clinical Guideline to appear in the *CJO* in as many issues, following on the Clinical Practice Guidelines for periodic eye examinations for children 0 - 5. Both documents are evidence-based and represent the best consensus among the experts involved in their writing. Reaching that consensus is not an easy task, particularly when different professional groups with somewhat disparate viewpoints are involved. How closely the individual clinician follows a particular Guideline is a matter of professional judgment and the patient's best interests should dictate how care is provided.

As I prepared to write this editorial, the latest coronavirus emerged from China and the news media were soon filled with advice about how to avoid it. References to the SARS outbreak of 2003 are common. Our medical and public health colleagues have plenty of guidelines to implement that were developed in the aftermath of SARS; it remains to be seen how well the guidelines are followed and whether they will be effective. Reporting of patients suspected of having the coronavirus is now mandatory for all Ontario health care providers, including optometrists.

As a profession we have looked forward to celebrating vision and vision care in the year 2020, and we certainly shall after the coronavirus crisis has passed. In the meantime, stay healthy and I'll see you in the next issue. ●

Notre article principal dans ce numéro est un résumé des lignes directrices de pratique de l'Association canadienne des optométristes sur la réadaptation optométrique de la basse vision. À mesure que la population du Canada vieillit, on s'attend à ce que le nombre de personnes touchées par des conditions qui entraînent une faible vision augmente considérablement et à ce que leur espérance de vie soit plus longue; par conséquent, ces personnes auront besoin de services de réadaptation de la basse vision beaucoup plus longtemps que les générations précédentes. Cela représente un grand défi non seulement pour les cliniciens individuels, mais aussi pour ceux qui planifient la logistique et le financement des soins de santé pour les aînés. Le guide de pratique clinique de 80 pages se trouve dans la section de la *Revue canadienne d'optométrie* (*RCO*) du système des revues à libre accès de l'Université de Waterloo à https://openjournals.uwaterloo.ca/index.php/cjo.

Il s'agit du deuxième guide de pratique clinique à figurer dans la *RCO* dans autant de numéros, après le « Guide de pratique clinique fondé sur des données probantes pour l'examen périodique de la vue chez les enfants de 0 à 5 ans au Canada ». Les deux documents sont fondés sur des données probantes et représentent le meilleur consensus parmi les experts qui ont participé à leur rédaction. Il n'est pas facile de parvenir à un consensus, surtout lorsque différents groupes professionnels ont des points de vue quelque peu différents. La mesure dans laquelle le clinicien suit une ligne directrice en particulier est une question de jugement professionnel, et l'intérêt supérieur du patient devrait dicter la façon dont les soins sont prodigués.

Alors que je me préparais à rédiger cet éditorial, le plus récent coronavirus est apparu en Chine, et les médias ont rapidement été remplis de conseils sur la façon de l'éviter. Les références à l'épidémie de SRAS de 2003 sont courantes. Nos collègues de la médecine et de la santé publique ont de nombreuses lignes directrices à mettre en œuvre qui ont été élaborées à la suite du SRAS; il reste à voir dans quelle mesure les lignes directrices sont suivies et si elles seront efficaces. La déclaration des patients soupçonnés d'avoir le coronavirus est maintenant obligatoire pour tous les fournisseurs de soins de santé de l'Ontario, y compris les optométristes.

En tant que profession, nous avons hâte de célébrer la vision et les soins de la vue en 2020, et nous le ferons certainement après la crise du coronavirus. Entre-temps, restez en santé, et je vous verrai dans le prochain numéro. ●

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2020 CAO Clinical Practice Guideline: Optometric Low Vision Rehabilitation Executive Summary

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PURPOSE

The purpose of the Low Vision Clinical Practice Guideline is to assist Canadian Optometrists in the provision of the best rehabilitative care for patients with low vision. The guideline is based on current available best evidence, interpreted by an expert panel. The writing group includes optometrists from academia and private practice, representing various regions across Canada. The guideline will aid optometrists to identify patients who require low vision rehabilitation and recommends appropriate assessment and management.

INTRODUCTION

The Problem

Currently, it is estimated that 0.95% of the Canadian population has visual impairment, including low vision and blindness.[1] The prevalence increases exponentially with age.[2] By the age of 75-84 years the percentage of those with visual impairment is approximately 6-8.9%.[1, 3] The Canadian population is aging;[4] by 2036 it is expected that 23-25% of the population will be comprised of older adults. There will, therefore, be an associated increase in people with visual impairment.[1, 3, 5, 6] It is predicted that the numbers of people with low vision will more than double in the next 30 years.[2] There is an urgent and increasing need for low vision rehabilitation (LVR) to help mitigate the impact of visual impairment.

The impact of visual impairment is wide-ranging and substantial. It is associated with disability (activity limitations) for visually intensive tasks (reading, writing), as well as mobility.[7] There are also deficiencies in performance of daily living tasks, such as personal care, shopping and meal preparation, compared to the general population, as well as compared to those living with other chronic conditions.[7-9] People with visual impairment are less likely to be employed, and have more social isolation and transportation difficulties and more risk of falls. They have higher risk of depression and other mental health difficulties, an overall reduction in quality of life and increased mortality.[8, 10-12]

Model of Low Vision Rehabilitation

Studies have shown that LVR is effective.[13, 14] This includes LVR for patients with mild vision loss,[15] and LVR provided by optometrists in both community and private practice settings.[15-17] Canadian optometrists are ideally qualified[18] to play a pivotal role in low vision provision, and are instrumental in full multidisciplinary/interdisciplinary settings, providing the initial assessment and interventions, plus devising a rehabilitation plan. Since Canada is large geographically and has sparse populations in rural areas, it is essential for equitable access that optometrists provide a key role in LVR.[19]

Level 1. LVR
This guideline adopts a three-tier model of LVR. It states that it is the responsibility and minimum standard of care expected of all optometrists to either directly provide LVR, or refer or recommend for LVR by a low vision optometrist prior to referral to other agencies. This is **Level 1 LVR** and should occur as soon as the patient experiences permanent low vision. LVR should be a parallel process to treatment for the eye condition when it is known that a degree of vision loss is irreversible.

Low vision assessment and rehabilitation should always be recommended for the following:

- A patient who has **low vision** which is defined as a **visual impairment** (measurable loss of vision) resulting in a **visual disability** (difficulty undertaking a task because of poor vision).

- To clarify, this includes all patients who have
  - An incurable disease or injury (ocular or systemic) for which available surgical or medical treatment has been undertaken, considered or is on-going
    AND
  - Reduced corrected vision (most commonly impairment of visual acuity, contrast sensitivity, or visual fields) compared to age norms
    AND
  - Difficulty with desired visual tasks despite optimum optical correction

- In terms of visual impairment, the levels at which vision loss is **likely** to cause a visual disability are (but not limited to) the following
  - VA 6/12 (20/40) or poorer
    OR
  - Central or paracentral scotoma or metamorphopsia
    OR
  - Peripheral field loss (hemianopia or quadrantanopia; less than 70 degrees[1] circular diameter total field)
    OR
  - Log CS < 1.4

Minimum additional assessment: In order to undertake an accurate referral, it is important to ascertain a patient's self-reported disabilities, functional vision and goals. An accurate refraction (ideally with a trial frame) and measurement of best corrected visual acuity are important. All optometrists should be willing and able to trial a higher reading addition (up to 4D). An assessment of contrast sensitivity and visual fields is highly recommended to complete the information required to make an accurate referral.

Beyond this requirement there are two levels at which optometrists may choose to provide LVR.

Level 2. Basic LVR
This level of LVR can be provided in an optometrist's office with a modest amount of equipment and optical devices and ideally with the support of a trained optometric technician/assistant or low vision therapist.

Patients who are likely to benefit are those with:

- VA from 6/12 to 6/21 inclusive *and/or* Log contrast sensitivity between 1.40 and 1.00

- No hemianopia or quadrantanopia, and circular visual field larger than 70 degrees

- No significant paracentral field loss which limits reading speed/visual function

Level 3. Comprehensive LVR
Patients with vision poorer than listed above for Level 2 Basic LVR are likely to require the full range of optical and electronic devices, and services as described in the Low Vision Clinical Practice Guide,[20] summarised below.

OPTOMETRIC LOW VISION REHABILITATION

Low vision rehabilitation starts with a full low vision assessment/evaluation which includes an extended evaluation of visual function and a review of ocular disease and systemic health conditions that may impact visual function (measured capability of the visual system) and functional vision (ability to undertake vision-related daily life tasks).[21] This results in the creation of an initial Low Vision Rehabilitation plan. Low Vision Rehabilitation management includes the assessment for and training with various optical and/or non-optical low vision aids and/or rehabilitation strategies directed towards the patient's specific needs, as well as supportive patient education and counselling. The result is the final rehabilitation plan, which is the final recommendations for the patient.

Low Vision Assessment

A comprehensive case history is conducted with emphasis on the patient's self-reported disabilities and goals, including a review of functional domains and covering activities of daily living, vocational/educational/avocational requirements and social activities, which may be impacted by the visual impairment. This is followed by prioritizing the goals with the patient. The case history should also investigate the effectiveness of current spectacles and devices, ocular, general health and family history, medication use, social history, any history of falls, the effects of glare and lighting, the stability of the ocular condition and the patient's own understanding of their ocular condition and its impact.

Trial frame refraction (objective followed by subjective) using lens changes based on the just noticeable difference, is an essential component of a low vision assessment. Often a significant VA improvement can be gained[22, 23] and it is important for the accurate assessment and dispensing of most optical devices that the correct refraction be in place. Habitual and corrected distance visual acuities preferably using visual acuity charts based on logMAR (log of the Minimum Angle of Resolution) principles and designed for low vision[24] and appropriate for the patient's level of vision and age.[24] Near visual acuity should ideally be measured with logMAR continuous text charts, and the viewing distance and threshold M print should be recorded. The impact of lighting on visual acuity should also be considered.

Contrast sensitivity is an important measure to understand a patient's visual function/disabilities and predict outcome with magnification.[25, 26] It is predictive of difficulty with of a wide range of other visual tasks (daily living skills, mobility, face discrimination, driving) and perceived disability.[7, 27-30] Poor CS is a also risk factor for falls.[31, 32] Visual field loss (central or peripheral must be considered and often measured. Additional assessments may be indicated such as colour vision and glare testing, to understand the loss of function due to the patient's ocular disease. Ocular health assessment allows the clinician to evaluate any progress in the disease and the contribution of multiple conditions causing low vision. Dilated fundus examination is not routinely included in a LV assessment as this is usually undertaken prior to the low vision assessment. It may be required, however, in cases where the symptoms, disabilities or other measurements do not align with the current diagnosis, or when there is no recent ocular health examination. Dilated fundus examination will normally require a separate appointment.

At the conclusion of the assessment, the optometrist is able to create a **Low Vision Rehabilitation Plan**. Many components can be implemented by the low vision optometrist, together with his/her optometric assistant. Implementation of the full plan may require referral to other service providers. The rehabilitation plan is revised after exploring and determining the appropriate management (including referrals) for the patient, described below.

Low Vision Management

The tools at the disposal of the LV clinician include optical, non-optical and electronic magnification, increased contrast, lighting control, minification, relocation of the object or image, training and adaptations. Patient and family education and referrals to other service providers are also important components.

Patients with **central vision loss** may be managed with optical and electronic magnification, as appropriate for distance, intermediate and near tasks. Optical magnification includes high adds and microscopes, hand and stand magnifiers and telemicroscopes for near or intermediate and telescopes for distance tasks. Many optical assistive devices may be customized to account for a patient's refractive error. Electronic magnification and mainstream technology accessibility options should also be considered for many patients. Electronic magnification is effective for patients with contrast sensitivity loss and/or large central scotomas, and includes hand-held, portable and desktop video magnifiers. Patients may often benefit from both optical and electronic magnification. Optical magnifiers tend to be used more frequently and for a variety of tasks while electronic magnification may allow reading for

longer duration, smaller print and be preferred for leisure reading.[33, 34] Accessibility features on current devices (mobile phone, tablet, laptop/desktop computers) includes text to speech, voice assist, talk-back, magnification and contrast/font/colour options.

Although there is limited evidence for the effectiveness of eccentric viewing training (EVT),[35] EVT still retains a place within the range of approaches for patients with central scotomas.

Reduced contrast sensitivity is the other main category of vision impairment causing disability alongside central vision impairment (VA loss) and visual field impairment. When CS is reduced to <1.40,[7, 29] the patient is likely to be experiencing some disabilities, such as issues with mobility and resolution tasks, but when CS is <1.00, visual performance is severely compromised, even with appropriate magnification. For example, reading is likely to be slow, even with the use of optical magnifiers.[25, 26, 36]

The approaches for contrast sensitivity loss include a) changing the patient's contrast sensitivity by manipulating the lighting, trialing filters or a typoscope or reversing contrast on electronic magnification or b) increasing the contrast of the task with electronic devices, environmental modifications, and using sight substitution methods e.g., voice output on a computer.

Management for **peripheral vision loss** includes use of prisms for hemianopia (Peli prisms or sector prisms), sector prisms or minifiers for constricted fields, visual search training, strategies to improve visual guidance while reading, and referral for orientation and mobility training.

Patients with **nystagmus** may benefit from yoked prisms and task positioning to enable the comfortable use of their null point. Contact lenses may improve VA for some patients with infantile nystagmus.[37, 38]

Lighting levels may significantly improve function for patients with visual impairment and should be explored. Non-selective and selective transmission filters can be of great benefit to many patients with low vision to control light levels and glare, and to optimise patient comfort. Short wave-length yellow tints are often subjectively beneficial to patients, although currently there is no objective evidence that they improve VA, CS or reading for people with visual impairment.[34] Tinted or iris imprint contact lenses may benefit patients with extreme photophobia.

For all devices and rehabilitation recommendations, the selection should involve a patient-centred decision process i.e. the best device for the task(s) as guided by the individual patient.

The optometrist should be able to recommend appropriate non-optical devices, such as large print books, clocks and watches, devices with auditory output, e.g. talking books and blood glucose monitors and tactile approaches such as markings for appliances. Communication and collaboration with other professionals in the rehabilitation team is important for patient success. Optometrists should refer when indicated for other services, such as orientation and mobility training, occupational therapy, low vision therapy, high technology assessments, social and community services, counselling, genetic counseling, vocational counselling, and surgical consultation when appropriate, e.g., for cataract, nystagmus, strabismus. When referring, it is recommended that the optometrist include his/her rehabilitation plan, including what interventions have been explored and implemented.

CONCLUSION

LVR requires a holistic approach to the patient, and the optometrist must be mindful of the emotional and psychological state of the patient. Interventions that are recommended should not only be task(s) specific, but also patient specific i.e. tailored for each particular patient's goals, requirements and limitations. LVR is an on-going process for most patients and follow-up is important as patients' acceptance level, activities and goals may change over time.

Optometrists are uniquely qualified to provide LVR, as they expertly refract, optimise visual function with spectacles and contact lenses, accurately assess visual function and understand the impact of ocular conditions, develop a vision rehabilitation plan, prescribe optical and non-optical, hand held and spectacle mounted devices, provide vision and assistive device training, advise about visual strategies and environmental modifications and co-ordinate with other services. ●

ENDNOTE

[1] This includes 60 degrees which is the level for funding in Quebec.

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Guide de pratique clinique 2020 de l'ACO : Réadaptation optométrique de la basse vision Sommaire

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OBJET

Le Guide de pratique clinique en basse vision a pour but d'aider les optométristes canadiens à fournir des soins optimaux de réadaptation aux patients atteints de déficience visuelle. Le Guide de pratique est fondé sur les meilleures données probantes disponibles, interprétées par un groupe d'experts. Le groupe de rédaction est composé d'optométristes du milieu universitaire et de la pratique privée, représentant diverses régions du Canada. Le Guide de pratique aidera les optométristes à identifier les patients qui ont besoin d'une réadaptation de la basse vision. Il recommande une évaluation et une prise en charge appropriées.

INTRODUCTION

Le problème

À l'heure actuelle, on estime que 0,95 % de la population canadienne souffre d'une déficience visuelle (basse vision, cécité, etc[1].) La prévalence augmente de façon exponentielle avec l'âge[2]. Entre 75 et 84 ans, le pourcentage de personnes souffrant d'une déficience visuelle atteint environ 6 à 8,9 %[1,3]. La population canadienne vieillit[4]; d'ici 2036, les personnes âgées devraient représenter de 23 à 25 % de la population. Il y aura donc une augmentation proportionnelle du nombre de personnes atteintes de déficience visuelle[1,3,5,6]. Le nombre de personnes ayant une basse vision devrait augmenter de plus du double au cours des 30 prochaines années[2]. Le besoin de réadaptation de la basse vision (RBV) est de plus en plus urgent pour réduire les effets de la déficience visuelle.

La déficience visuelle entraîne des répercussions considérables et de grande envergure. Elle est associée à une incapacité (limitations d'activité) pour la mobilité et pour les tâches visuellement intensives (lecture, écriture)[7]. Elle est également cause de défaillance dans l'exécution des tâches de la vie quotidienne, comme les soins personnels, les courses et la préparation des repas, par rapport à la population générale et aux personnes atteintes d'autres maladies chroniques[7-9]. Les personnes souffrant d'une déficience visuelle sont moins susceptibles d'avoir un emploi et plus isolées sur le plan social, elles ont aussi plus de problèmes de transport et risquent davantage de faire une chute. Elles sont plus sujettes à la dépression, à d'autres problèmes de santé mentale, à une réduction globale de leur qualité de vie et à un risque plus élevé de mortalité[8,10-12].

Modèle de réadaptation de la basse vision

Des études ont prouvé l'efficacité de la RBV[13,14] fournie par les optométristes en milieu communautaire ou en privé[15-17], y compris pour les patients présentant une perte de vision mineure[15]. Les optométristes canadiens sont idéalement qualifiés[18] pour jouer un rôle déterminant dans la prestation de traitements de la basse vision, et ils exercent dans des contextes multidisciplinaires ou interdisciplinaires intégraux où ils effectuent l'évaluation initiale, réalisent les premières interventions et mettent au point un plan de réadaptation. Vu l'immense superficie du Canada et la dispersion de sa population rurale, il est essentiel pour assurer un accès équitable aux services que les optométristes jouent un rôle clé dans la RBV[19].

Niveau 1. RBV
Ce Guide de pratique clinique adopte un modèle à trois niveaux de RBV. Il énonce qu'à titre de norme minimale de soins, il incombe à tous les optométristes de fournir directement des services de RBV ou de recommander de tels services par un optométriste spécialiste de la basse vision, avant tout aiguillage vers d'autres organismes. Ceci constitue une **RBV de niveau 1** qui devrait avoir lieu dès que le patient souffre d'une déficience visuelle permanente. Lorsqu'il est avéré qu'un degré de perte de vision est irréversible, la RBV devrait constituer un processus parallèle au traitement de la maladie oculaire.

L'évaluation et la réadaptation en basse vision devraient toujours être recommandées dans les cas suivants :

- Un patient ayant une **basse vision** qui est définie comme étant une **déficience visuelle** (perte mesurable de la vision) entraînant une **incapacité visuelle** (difficulté à entreprendre une tâche en raison d'une mauvaise vision).

- À titre de précision, sont comprises toutes les pathologies suivantes :
  - une maladie ou blessure incurable (oculaire ou systémique) pour laquelle un traitement chirurgical ou médical a été entrepris, envisagé ou est en cours;
    ET
  - une réduction de la vision corrigée (le plus souvent atteinte de l'acuité visuelle, de la sensibilité différentielle ou des champs visuels) par rapport aux normes de l'âge;
    ET
  - une difficulté à accomplir les tâches visuelles souhaitées en dépit d'une correction visuelle optimale.

- En ce qui a trait à la déficience visuelle, les niveaux auxquels la perte de vision est **susceptible** de causer une incapacité visuelle sont les suivants, sans toutefois s'y limiter :
  - acuité visuelle (AC) de 6/12 (20/40) ou inférieure;
    OU
  - scotome central ou paracentral ou métamorphopsie;
    OU
  - perte périphérique de champ (hémianopsie ou hémianopsie en quadrant; champ visuel circulaire total de moins de 70 degrés[1]);
    OU
  - Log CS < 1,4

Évaluation supplémentaire minimale : Afin d'assurer un aiguillage approprié, il est important de vérifier les incapacités autodéclarées d'un patient, sa vision fonctionnelle et ses objectifs. Il est important que la réfraction soit exacte (idéalement avec des montures d'essai) et que la mesure de l'acuité visuelle corrigée soit la meilleure possible. Tous les optométristes devraient être disposés et aptes à tester des additions plus élevées pour la lecture (jusqu'à 4D). Il est fortement recommandé de procéder à une évaluation des champs visuels et de la sensibilité différentielle pour recueillir les renseignements nécessaires à un aiguillage approprié.

Au-delà de cette exigence, il y a deux niveaux auxquels les optométristes peuvent choisir de fournir des services de RBV.

Niveau 2. RBV de base
Ce niveau de RBV peut être fourni dans le bureau d'un optométriste avec une gamme réduite d'équipements et de dispositifs optiques et, idéalement, avec le soutien d'un technicien ou aide-optométriste qualifié, ou d'un thérapeute en basse vision.

Les patients qui sont susceptibles d'en bénéficier sont ceux qui présentent les symptômes suivants :

- AV de 6/12 à 6/21 inclusivement *et/ou* sensibilité différentielle Log entre 1,40 et 1,00;

- aucune hémianopsie ou hémianopsie en quadrant, et champ visuel circulaire de plus de 70 degrés;

- aucune perte importante de champ périphérique qui limite la vitesse de lecture ou la fonction visuelle.

Niveau 3. RBV global
Les patients dont la vision est plus faible que celle indiquée ci-dessus pour le RBV de base de niveau 2 auront probablement besoin de toute la gamme d'appareils optiques et électroniques, ainsi que des services décrits dans le Guide de pratique clinique de basse vision[20], résumé ci-dessous.

RÉADAPTATION OPTOMÉTRIQUE DE LA BASSE VISION

La réadaptation de la basse vision commence par une évaluation complète et approfondie de la fonction visuelle et un examen des maladies oculaires et des problèmes de santé systémiques qui peuvent avoir un effet sur cette fonction (capacité mesurée du système visuel) et sur la vision fonctionnelle (capacité d'entreprendre les tâches de la vie quotidienne liées à la vision[21]). Il en résulte la création d'un plan initial de réadaptation de la basse vision. La gestion de la réadaptation de la basse vision comprend l'évaluation du patient et la formation au moyen de diverses aides optiques ou non optiques à la basse vision, de diverses stratégies de réadaptation axées sur les besoins particuliers, et d'éducation et de conseils à l'intention des patients. Le résultat est le plan de réadaptation qui contient les recommandations finales pour le patient.

Évaluation de la basse vision

Un historique complet du cas est effectué avec attention particulière sur les incapacités et les objectifs autodéclarés du patient, notamment un examen des domaines fonctionnels, activités de la vie quotidienne, activités sociales, et exigences professionnelles, éducatives ou de loisirs qui peuvent être touchés par la déficience visuelle. Il faut ensuite établir l'ordre de priorité des objectifs avec le patient. L'étude de cas doit également porter sur l'efficacité des lunettes et des appareils actuels, les antécédents oculaires, familiaux, sociaux, de santé en général, de consommation de médicaments et de chutes, ainsi que sur les effets de l'éblouissement et de l'éclairage, la stabilité de l'état oculaire et la compréhension du patient lui-même de son état de santé oculaire et des conséquences qui en découlent.

La réfraction avec les montures d'essai (objective suivie de subjective) à l'aide de changements de lentille fondés sur le seuil différentiel est un élément essentiel de l'évaluation de la basse vision. Il est souvent possible d'obtenir une amélioration importante de l'AV[22,23] et le discernement de la réfraction exacte est un facteur essentiel pour l'exactitude de l'évaluation et de la prescription de la plupart des appareils optiques. Les acuités visuelles à distance habituelles et corrigées, de préférence à l'aide de tableaux d'acuité visuelle basés sur les principes du logMAR (l'angle minimum de résolution) et conçus pour la basse vision[24] et adaptés au niveau de vision et à l'âge du patient[24]. Il faudrait idéalement mesurer l'acuité visuelle de près à l'aide des tableaux à texte continu du logMAR, et enregistrer la vision à distance et la lecture de mots imprimés. Il faudrait également tenir compte des effets de l'éclairage sur l'acuité visuelle.

La sensibilité différentielle est une mesure importante pour évaluer la fonction visuelle ou une incapacité du patient et pour prédire le résultat avec un grossissement[25,26]. Elle permet de prédire les difficultés d'une vaste gamme d'autres tâches visuelles (aptitudes à la vie quotidienne, mobilité, discrimination du visage, conduite) et incapacité perçue[7,27-30]. Une faible sensibilité différentielle est également un facteur de risque de chute[31,32]. Il faut tenir compte de la perte du champ visuel (central ou périphérique) et la mesurer régulièrement. Pour déterminer la perte de fonction due à la maladie oculaire du patient, il peut être indiqué de procéder à des examens supplémentaires, tels que la vision des couleurs et les tests d'éblouissement. L'évaluation de la santé oculaire permet au clinicien de constater tout progrès de la maladie et la contribution de multiples causes à une basse vision. L'examen du fond de l'œil dilaté n'est pas systématiquement inclus dans une évaluation de la basse vision, car il est habituellement effectué auparavant. Par contre, il peut être nécessaire dans les cas où les symptômes, les incapacités ou d'autres mesures ne correspondent pas au diagnostic actuel ou en l'absence d'examen récent de la santé oculaire. Il faudra habituellement un rendez-vous distinct pour l'examen du fond de l'œil dilaté.

À la fin de l'évaluation, l'optométriste est en mesure de créer un **plan de réadaptation de la basse vision**. L'optométriste spécialiste de la basse vision et son assistant peuvent mettre en place de nombreuses composantes. La mise en œuvre du plan complet peut nécessiter l'aiguillage vers d'autres fournisseurs de services. Après une détermination de la prise en charge appropriée du patient (y compris les aiguillages), décrite ci-dessous, le plan de réadaptation fait l'objet d'une révision.

Gestion de la basse vision

Les outils à la disposition du clinicien en basse vision comprennent le grossissement optique, non optique et électronique, l'augmentation du contraste, le contrôle de l'éclairage, la minimisation, le déplacement de l'objet ou de l'image, la formation et les adaptations. Constituent d'autres éléments importants l'éducation des patients et de leur famille et l'aiguillage vers d'autres fournisseurs de services.

Les patients atteints d'une **perte de vision centrale** peuvent être pris en charge par grossissement optique et électronique, selon les besoins pour les tâches éloignées, semi-éloignées et rapprochées. Le grossissement optique pour les besoins relatifs aux tâches proches ou semi-éloignées se fait au moyen de prismes élevés, de microscopes, de télémicroscopes et de loupes à main et sur pied, et pour les tâches à distance, au moyen de télescopes. De nombreux appareils d'assistance optique peuvent être personnalisés pour tenir compte de l'erreur de réfraction d'un patient. Il faudrait également envisager pour de nombreux patients le grossissement électronique et les options d'accessibilité de la technologie traditionnelle. Le grossissement électronique se fait au moyen de loupes à main, portables et de

bureau. Il est efficace pour les patients qui présentent une perte de sensibilité différentielle ou d'importants scotomes centraux. Les patients peuvent souvent bénéficier à la fois du grossissement optique et électronique. Les patients ont tendance à utiliser plus fréquemment les loupes optiques pour une variété de tâches, et à préférer le grossissement électronique pour lire plus longtemps, lire des caractères plus petits et pour leur lecture de loisir[33,34]. Les fonctions d'accessibilité des appareils actuels (téléphone mobile, tablette, ordinateur portatif ou de bureau) comprennent des options de texte, d'assistance vocale, de grossissement, de contraste, de police et de couleur.

Bien qu'il y ait peu de preuves de l'efficacité de l'entraînement à la vision excentrique[35], elle continue de faire partie de l'éventail des solutions pour les patients atteints de scotomes centraux.

La **sensibilité différentielle réduite** est l'autre principale catégorie de troubles de la vision causant une incapacité parallèle à la perte de la vision centrale (perte d'AV) et des troubles du champ visuel. Lorsque la sensibilité différentielle est réduite à moins de 1,40[7,29], le patient risque d'avoir certaines incapacités, comme des problèmes de mobilité et de résolution, mais lorsqu'elle est inférieure à 1,00, la performance visuelle est gravement compromise, même avec un grossissement approprié. Par exemple, la lecture est probablement lente, même avec l'aide de loupes optiques[25,26,36].

Les approches pour la perte de sensibilité différentielle comprennent a) la modification de la sensibilité différentielle du patient par une manipulation de l'éclairage, l'essai de filtres ou d'un typoscope, ou l'inversion du contraste sur un grossissement électronique ou b) l'augmentation du contraste de la tâche avec des dispositifs électroniques, des modifications de l'environnement et l'utilisation de méthodes de substitution de la vue, par exemple, la sortie vocale sur un ordinateur.

La gestion de la **perte de vision périphérique** comprend l'utilisation de prismes pour l'hémianopsie (des prismes sectoriels ou des prismes de Peli), de prismes sectoriels ou de minimiseurs pour les champs resserrés, la formation à la recherche visuelle, les stratégies pour améliorer l'orientation visuelle pendant la lecture, l'aiguillage vers une orientation et une formation sur la mobilité.

Les patients atteints de **nystagmus** peuvent bénéficier de yoked prisms et du positionnement des tâches pour permettre l'utilisation confortable de leur point nul. Il est possible d'améliorer l'AV de certains patients atteints de nystagmus infantile au moyen de lentilles cornéennes[37,38].

Les niveaux d'éclairage peuvent améliorer considérablement la fonction des patients ayant une déficience visuelle et il convient d'en faire l'essai. Il peut être très avantageux pour de nombreux patients d'utiliser les filtres de transmission sélectifs et non sélectifs pour contrôler les niveaux de lumière et d'éblouissement et d'optimiser leur confort. Les teintes jaunes de courte longueur d'onde offrent souvent des avantages subjectifs pour les patients, bien qu'il n'y ait actuellement aucune preuve objective qu'elles améliorent l'AV, la lecture ou la sensibilité différentielle chez les personnes ayant une déficience visuelle[34]. Les lentilles cornéennes teintées ou irisées peuvent être bénéfiques pour les patients souffrant de photophobie extrême.

Pour tous les appareils et toutes les recommandations de réadaptation, la sélection devrait suivre un processus décisionnel axé sur le patient, c.-à-d. le meilleur dispositif pour la ou les tâches du patient, en fonction de sa propre appréciation.

L'optométriste devrait être en mesure de recommander des appareils non optiques appropriés, comme des livres, horloges et montres à gros caractères, des appareils avec sortie auditive, p. ex., livres et moniteurs de glycémie sonores, et des approches tactiles comme le marquage des appareils. Il est important pour le progrès des patients de communiquer et de collaborer avec les autres professionnels de l'équipe de réadaptation. Lorsque la situation le justifie, l'optométriste devrat aiguiller le patient vers les autres services comme la formation à l'orientation et la mobilité, l'ergothérapie, la thérapie contre la basse vision, les évaluations de haute technologie, les services sociaux et communautaires, le counseling, les services de consultation en génétique, les conseils professionnels et la consultation chirurgicale au besoin, p. ex., cataracte, nystagmus, strabisme. Au moment de l'aiguillage, il est recommandé que l'optométriste inclue son plan de réadaptation, ainsi que les interventions qui ont été envisagées et mises en œuvre.

CONCLUSION

La RBV nécessite une approche holistique à l'égard du patient, et l'optométriste doit être conscient de son état émotionnel et psychologique. Les interventions qui sont recommandées doivent être particulières non seulement aux tâches, mais aussi au patient lui-même, c.-à-d. adaptées à ses objectifs, ses exigences et ses limites. La RBV est un processus continu pour la plupart des patients et il est important d'assurer un suivi, car le niveau d'acceptation, les activités et les objectifs des patients peuvent changer au fil du temps.

Les optométristes sont particulièrement qualifiés pour fournir de la RBV, car ils réfractent de façon experte, optimisent la fonction visuelle avec des lunettes et des lentilles cornéennes, évaluent avec précision la fonction visuelle, comprennent les effets des problèmes oculaires, élaborent un plan de réadaptation visuelle, prescrivent des appareils optiques et non optiques, portatifs et montés sur des lunettes, fournissent une formation sur la vision et les appareils fonctionnels, donnent des conseils sur les stratégies visuelles et les modifications de l'environnement et coordonnent leurs interventions avec les autres services. ●

NOTE EN FIN DE TEXTE

[1] Cela comprend 60 degrés, ce qui est le niveau pour obtenir un financement au Québec.

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2020 CAO Clinical Practice Guideline: Optometric Low Vision Rehabilitation (Full Guidelines)

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CONTENTS

1. ABSTRACT

The purpose of the Low Vision Clinical Practice Guideline for Canadian Optometrists is to assist them in providing the best level of care in the management of patients with low vision. The guideline is based on current best evidence regarding optometric low vision rehabilitation as interpreted by an expert panel. The writing group includes optometrists from academia and private practice, representing various regions across Canada. This guideline will aid optometrists in identifying patients who require low vision rehabilitation and recommends appropriate assessment and management.

As primary eye care providers, optometrists are optimally trained and qualified to identify and manage patients who would benefit from low vision rehabilitation. Optometrists, based on their geographical presence in local communities, are also well positioned to provide initial rehabilitation in a timely, effective manner, and to initiate referral for more comprehensive rehabilitation if required.

2. INTRODUCTION

2.1 DEFINITIONS

Table 1: *Definitions and abbreviations*

CPS	Critical print size. The smallest print at which maximum[1,2] or near maximum reading speed is attained[3]. CPS is distance dependent, which must be specified. It can be written as a visual acuity fraction (distance as numerator and M print as denominator).
CS	Contrast sensitivity
EVP	Equivalent Viewing Power. This is used to describe the equivalent power of optical devices and systems, such as stand magnifiers (which are used in combination with a reading add) or telemicroscopes. It is the power of a microscope which would give the same equivalent magnification or power.
logMAR	Logarithm of the Minimum Angle of Resolution
LV	Low Vision. An incurable visual impairment which cannot be sufficiently improved with optical correction and which interferes with activities.[4-6]
LVR	Low vision rehabilitation (low vision services, low vision intervention, low vision care) includes low vision assessment and may include the provision of low vision assistive devices, environmental/lifestyle modifications, education, training and alternative techniques.
PRL	Preferred retinal locus. One or more regions of functioning retina consistently used for fixation instead of the fovea in an individual with bilateral dense central scotomas. The PRL may be used for attentional deployment and as the oculomotor reference.[7]
VA	Visual acuity
Handicap or participation restriction	A limitation in involvement in life, a psychosocial disadvantage that occurs because of the disability[5,8]
Visual disorder	Any deviation from the normal structure or function of the eye or visual system due to disease, trauma or congenital anomaly[5,8]
Visual disability/ activity limitation	A reduced ability to perform a desired task (for that individual) because of the visual impairment[5,8]
Visual impairment	A measurable chronic reduction of vision compared to the normal age-related values. It is assumed that all optical, surgical or medical treatment has been undertaken or considered[5,8]

2.1.1 LOW VISION

The World Health Organization (WHO) provided a classification system to address the consequences of disease. They proposed that **disease** causes an **impairment** in structure or function. This, in turn, creates a **disability/activity limitation** wherein the individual is unable to complete a particular task in the usual manner, as a result of the impact of the impairment. A **handicap/participation restriction** occurs when the individual's interaction with their environment is affected as a result of the disability.[8]

For clinical purposes the determination of low vision is ideally determined by the presence of incurable disease that impairs visual function. However, research and legal definitions often define low vision on the degree of visual acuity impairment. The WHO considers an individual as having low vision when best corrected visual acuity (VA) is poorer than 6/18 (20/60) to 6/120 (20/400). A person with VA poorer than 6/120 or with a visual field of less than 10 degrees is considered legally blind.[9] The North American classification defines legal blindness as 6/60 or poorer, or a visual field of 20 degrees or less, and recognizes that low vision occurs with less severe acuity impairment, defining low vision as worse than 6/12 (<20/40).[10] This level is identified by other authors as a level at which disability typically begins.[4,5,11] In Canada, definitions for eligibility for services and assistive plans are typically based on specific levels of visual acuity or visual field loss (See Appendix A for details regarding eligibility for low vision rehabilitation and devices according to province).

This guideline adopts a functional or disability-based definition of low vision, which is patient-centred, and seeks to account for the full scope of a patient's visual impairment and their individual disabilities. **Low vision is defined as occurring when a visual impairment results in a person not being able to perform his or her desired visual tasks.** It is based on the best monocular or binocular performance which is expected to be long-standing. Typically, low vision is either due to loss of visual acuity, visual fields or contrast sensitivity, or a combination of these.[4,5] For visual field impairment loss, disability starts when the visual fields is <70° circular diameter or equivalent.[12] For contrast sensitivity, a disability starts when performance on a letter test of contrast sensitivity drops to Log CS of <1.40.[13-15] Nevertheless, neither of these criteria are strict cut-offs; they are intended as general indications only.

2.1.2 LOW VISION REHABILITATION

Low vision rehabilitation (LVR) includes visual assessment specific to visual impairment and may include the provision of low vision assistive devices, environmental/lifestyle modifications, education, training and alternative techniques. The purpose of LVR is to help the individual with low vision to achieve their personal visual goals, attain maximum function, achieve their desired level of independence, and attain a safe and satisfying lifestyle. Synonyms include low vision services, low vision intervention, and low vision care.

2.2 EPIDEMIOLOGY

To date, there is no comprehensive population based study of low vision prevalence in Canada. The studies which do exist include correctable forms of impairment such as refractive error and/or cataract. Using a definition which includes individuals with cataracts, Maberley et al. provided useful national prevalence data based on extrapolations from clinical records of best-corrected acuity and visual field status for the better-seeing eye in a medium sized Canadian city. They estimated that 0.39% of the total population has visual impairment (including low vision and blindness) according to the WHO classification (<6/21 [<20/70]) which increased to 0.95% utilizing the North American definition (<6/12 [<20/40]). Using the latter definition, the prevalence rises to 8.9% of people aged 75-84 years and 18% of those aged 85+.[10] These estimates include about 30% of all patients having impaired vision due to cataract or complications of cataract as the primary etiology, suggesting possible over-estimation. A more recent study reported that 5.7% of Canadian adults aged 45-84 years have some form of reduced vision. This percentage increased from 2.7% in 45-54 year olds to 15.6% in 75-84 year olds.[16] Notably, uncorrected refractive error was thought to be the cause of reduced vision for 64-80% of participants, while self-reported cataract was present for 5.6%.[16]

More comprehensive population studies have been conducted in other developed countries. The studies focused on middle aged and older adults, and most used a definition of best-corrected VA of 6/12 (20/40) or worse. These include the Beaver Dam study (5.9%),[17] the Salisbury Eye Evaluation study (3.7%),[18] and the Blue Mountain study (4.7%).[19] These studies all demonstrated an increased prevalence of visual impairment with age. Chan et al. showed that visual impairment rises exponentially with age.[20] They modelled the prevalence of visual impairment in the US over the next 30 years and predicted a similar prevalence for best corrected VA worse than 6/12 of 3.9% for ≥45 year olds, resulting in a doubling of the number of people with visual impairment in that time. This age dependence is a concern, as the Canadian population is aging, with expectations that approximately 23-25% of the population will

be comprised of older adults by 2036.[21] The rise in diabetes rates with age, and the associated visual impairment due to diabetic retinopathy, is an additional concern. Between the ages of 75 and 79, 23.1% women and 28.5% men have diabetes.[22] The rise in total numbers of new cases of visual impairment indicates a substantial increase in the need for low vision rehabilitation services over the next 30 years.[20]

The distribution of severity of visual impairment among individuals is relevant in anticipating the scope of disability and the corresponding demand for rehabilitation. A large portion of the visually impaired population have mild or moderate visual impairment. In the population study by Attebo et al., 72% of older adults with visual impairment had mild visual impairment (6/12 to 6/18)[19] while Chan et al. reported that 60% of cases were mild LV, 23% were moderate (<6/18 to better than 6/60) and 17% were ≤6/60.[20] Goldstein et al. reported that 37% of patients served by outpatient low vision services had mild LV (VA equal or better than 6/18 [20/60]) while a further 38% had moderate visual impairment (6/18-<6/60 [20/60-<20/200]).[23] Contrast sensitivity deficits were classified as mild in 24% and moderate in 43% . This predominance of mild visual impairment would be consistent with the expectation that the needs of most individuals with impaired vision could be met with more modest interventions, such as a thorough optometric low vision rehabilitation rather than requiring more comprehensive, multidisciplinary approaches.[24]

2.3 IMPACT OF VISUAL IMPAIRMENT

The impact of visual impairment can be substantial. With the progression of the disease, and subsequent impairment, there is an associated increased difficulty with visually intensive tasks such as reading and mobility.[15] There are also deficiencies in performance of daily living tasks, such as personal care, shopping and meal preparation. This is true compared to the general population, as well as compared to those living with other chronic conditions.[25] Often, those with suboptimal vision will fail to meet the vision driving standard resulting in loss of their driver's license. This further exacerbates other negative effects of impairment, such as social isolation, as driving is typically the desired method of transportation.[26] People with visual impairment are more likely to discontinue their education or take longer to achieve their educational goals.[27] Only 35-45% of visually impaired people are employed,[27-29] and this is lower still for women (only 24.5% being employed), younger people and those with diabetes. Those with visual impairment are more likely to experience multiple falls.[30,31] For visual acuity <20/30 the prevalence ratio is 1.9 and for 5 or more missing points on the visual field the prevalence ratio is 1.5.[32] These factors and others contribute to an overall decrease in quality of life, and negatively affect mental health, leading to higher levels of depression.[33] People with visual impairment due to diabetes are especially at risk for difficulty adjusting to vision loss.[33] Older adults with visual impairment are 2x more likely to have depression and have difficulty with emotional adjustment and isolation.[34] It is noteworthy that there is no clear association between the level of visual impairment and the severity of depression,[35] although better adjustment to vision loss is documented for people with better VA.[33] There are increased rates of suicide and mortality among people with visual impairment; even mild visual impairment increases mortality by more than 2 times.[36-38]

2.4 THE NEED FOR LOW VISION REHABILITATION AND THE ROLE OF OPTOMETRISTS

Despite the documented effectiveness of LVR (see Appendix B), many people with low vision are not accessing low vision rehabilitation services. This is either because they are not referred, are not aware of such services, or because there are barriers that dissuade them from attending.[39] In Quebec, 75% of community-dwelling people aged 65 years+ with visual impairment had not utilised low vision services[40] while 67% of ophthalmology clinic patients with low vision had not heard of, or been referred to, LVR.[41] In Ontario, 74% of community dwelling adults were unaware of low vision services[42] and 50% of hospital patients with low vision were not referred for LVR.[43] Those less likely to be referred include those with visual disorders other than age-related macular degeneration, less education, more recent vision loss, and reduced VA that doesn't meet legal blindness requirements. Those who live alone and certain ethnic groups such as African Canadians are also less likely to be referred.[41] As primary vision care providers, all optometrists have a vital role to play in identifying patients who could benefit from LVR and ensuring that patients who require LVR are informed about low vision rehabilitation services. Optometrists may choose to provide that service themselves or refer accordingly. It is known that low vision rehabilitation is often more effective when provided at the early stages of visual impairment. It is important to address visual disabilities early, so that the patient does not experience unnecessary years of disability and participation restriction. Therefore, referral for, or provision of, LVR should be as soon as the patient experiences permanent low vision. LVR should be a parallel process to treatment when it is known that vision loss is irreversible.

2.5 THE EFFECTIVENESS OF LOW VISION REHABILITATION

Appendix B summarises systematic reviews regarding the effectiveness of various low vision rehabilitation interventions. According to the systematic review of Binns et al. there is good evidence that low vision rehabilitation, as a whole, is effective. Over the years, the question has been approached using different outcomes and study designs.

Cohort studies have provided very good evidence of the effectiveness of low vision rehabilitation as demonstrated by: improvements in clinical measures of ability to read smaller print, reading speed or reading duration with magnification devices;[44-46] the value that patients place on their devices and their continued use of devices (reported between 67-99%); patients' reported satisfaction with low vision services (between 83 and 98%);[46-50] patients' self-reported functional ability[46] and improved quality of life.[51-56]

Randomised clinical trials give a stronger level of evidence and have also indicated that low vision services are effective. One of the most comprehensive studies to date is the LOVIT randomised clinical trial which showed that full multidisciplinary LVR as offered by the U.S. Department of Veterans Affairs is effective compared to delayed intervention.[57] There were significant improvements in all areas of visual function for the treatment group. Significant benefit has also been shown for a broader demographic of patients attending out-patient low vision clinics, although with a smaller effect size.[58]

2.5.1 LOW VISION REHABILITATION PROVIDED BY OPTOMETRISTS

Low vision rehabilitation, primarily provided by optometrists in the community, has also been shown to be effective.[59] Patients valued the optometric low vision service which they received in university-based or community-based low vision clinics and continued to use their assistive devices.[49,50,60] The improvements in quality of life were similar between individuals experiencing optometric low vision rehabilitation and multidisciplinary services.[55] The recent LOVIT II study (a randomised clinical trial) showed no differences in visual function outcomes between basic and fully comprehensive services for people with relatively mild visual impairment (in the range of VA 6/15 to 6/18 [20/50 to 20/60]).[24]

2.6 MODELS OF LOW VISION REHABILITATION

Since the early 20th century, a variety of models of low vision care provision have developed including in-patient, out-patient, hospital-based, community-based, individual and group programs.[46,61] In Scandinavia[62] and Australia[63] the concept of multidisciplinary/interdisciplinary clinics that included vision therapists was developed. A full multidisciplinary team may include optometrists, ophthalmologists, opticians, social workers, low vision therapists, high technology assessors, counsellors, orientation and mobility specialists and occupational therapists. These clinics were centred in the community and were shown to provide beneficial results. Despite little evidence regarding the relative effectiveness of different models,[55,58,64,65] the multidisciplinary/interdisciplinary model of comprehensive low vision rehabilitation has become generally accepted as the "gold standard" for people with more severe visual impairment and more complex needs.[61,66-70]

It is recognized, however, that not all people with visual impairment require full multidisciplinary intervention. The WHO reported that approximately 80% of people with less severe levels of low vision can benefit from intervention at a basic level.[61,66] Therefore, the WHO recommends a three tier model of low vision provision.[61,66] In Wales, this concept was shown to be effective[48], with local optometrists providing initial services and linking with other local community-based professionals and voluntary organisations to provide some level of multidisciplinary support when necessary.[50] This service model improved accessibility to low vision rehabilitation resulting in more people receiving LV services and decreased waiting times and travel distances for patients. A similar model with optometrists providing initial, basic low vision rehabilitation has been suggested for Canada[71] and Australia[72], both large geographic areas with sparse populations in many regions. For equitable patient access to rehabilitation, optometrists are ideally qualified[73] and situated to be key players in basic low vision provision. They are also instrumental in full multidisciplinary/interdisciplinary settings, providing the initial assessment and interventions, plus devising a rehabilitation plan.[74] The model proposed for Canada is a three tiered model[71]. It is described in Appendix C and includes the following levels:

1. Screening and recognition of a potential patient with low vision followed by appropriate triage,
2. LVR of a patient with mild visual impairment/disability,
3. Comprehensive LVR for patients with more severe visual impairment and greater disabilities whose rehabilitation requires collaboration with other professionals.

Such a model, with two levels of LVR, has been adopted in Ontario by the Eye Health Council of Ontario.[75] This Clinical Practice Guideline is intended to assist optometrists who provide LVR at Levels 2 or 3.

3. OPTOMETRIC LOW VISION REHABILITATION

LVR includes Low Vision Assessment and Low Vision Management. It starts with a full low vision assessment which is an extended evaluation of visual function and a review of ocular disease and systemic health conditions that may impact visual function and functional vision. Visual function is the measured capability of the visual system and functional vision relates to the ability of the person to undertake vision-related tasks.[76] The low vision assessment results in the creation of an *initial* Low Vision Rehabilitation plan for low vision management. Low Vision Rehabilitation management includes the assessment for, and training with, various optical and/or non-optical low vision aids and/or rehabilitation strategies directed towards the patient's specific needs, as well as supportive patient education and counselling. The result is the final rehabilitation plan, which is the final recommendations for the patient.

3.1 LOW VISION REHABILITATION – ASSESSMENT

3.1.1 CASE HISTORY

The low vision assessment starts with a comprehensive case history. The comprehensive case history should be targeted towards the patient's self-reported disabilities and goals, but should also review the functional domains which might be impacted by the visual impairment, including activities of daily living, social activities, recreational activities and vocational/educational requirements.[77] It may be useful to ask the patient to describe their typical day and then ask how they manage each task. For a complete Activity Inventory of tasks and subtasks see Massof.[78]

Because of the typical variety and number of goals, it may not be possible to address all in one session, so prioritizing the goals with the patient is often helpful at this stage. Other important components of the case history are an evaluation of the effectiveness of current spectacles, low vision devices, and other adaptive strategies for a range of tasks. Discussing the effects of glare and lighting on their daily activities is also helpful.

Ocular, general health and family history and current medications should be reviewed, including the stability of the ocular condition and any current or future treatments. Additionally, the case history should explore the patient's understanding and perception of his/her eye condition and its impact on functional vision and the likelihood of progression. It is also important to determine any recent history of falls.

The patient's social history should be discussed, including living arrangements, mobility and use of support and community services. The optometrist should be cognisant of the likelihood of depression among people with low vision, and a depression screener such as the PHQ-2 can be administered (Table 2). For a child patient it is important to determine what school support services are in place.

Some additional information may be gathered from reports and referrals. The use of an intake questionnaire is useful to gather most of this information in advance and can help the patient identify their primary difficulties and goals.

For a list of case history components, refer to the American Optometric Association Guideline, Care of the Patient with Visual Impairment, Appendix Table 3.[68]

Table 2: *PHQ-2*[79]

During the last 2 weeks, how often have you been bothered by the following?	Not at all	Several days	More than half the days	Nearly every day	Score for question
Little interest or pleasure in doing things	0	1	2	3	
2. Feeling down, depressed or hopeless	0	1	2	3	
Total score (a score of 3 or more is positive for depression)					

3.1.2 VISUAL ACUITY

Distance visual acuity: The assessment usually continues with the measurement of entering distance VA. Printed charts are recommended for patients with low vision, as testing distances can be easily varied depending on acuity level and the illumination can be changed without changing the contrast. Charts based on a logMAR design such as the Early Treatment Diabetic Retinopathy Study (ETDRS) and Bailey-Lovie charts are preferred, because they have equal numbers of letters per line (usually five), equivalent spacing between

letters and lines to control crowding, and follow a logarithmic progression. This means that visual acuity measurements are consistent with different testing distances.[80-82] The use of multiple letters per row gives useful information regarding scotoma position, eccentric viewing, fixation stability and eye movement control. Other charts, such as the Feinbloom, Feinbloom PV numbers, or Lea numbers are useful as they are more portable and enable the measurement of very low levels of acuity. However, they are not designed in a fully logarithmic progression for letter size and spacing and do not have equal numbers of numbers per line over the whole chart.[83] For patients with very low visual acuity, the Berkeley Rudimentary Vision Test quantifies VA as low as 6/4800 (20/16,000).[84] Electronic versions of VA charts are also available, for example, the Freiburg Acuity Test, which can also measure VA to low levels.[85] Computerised charts are likely to become more widely used, and will allow more diversity of variables such as optotype, spatial arrangement and contrast, but careful documentation is important for standardisation and comparison.[86]

For children, charts using the Lea or Patti Pics symbols are available in a variety of formats in a logarithmic scale. Preferential looking with Teller cards or Cardiff cards can be used to measure VA for infants or patients with developmental delays. Visually evoked potentials can be used for those who cannot respond to any of these tests. Care must be taken in the interpretation of VA measured either with preferential looking or visually evoked potentials, since these measure resolution VA which is not equivalent to VA measured with letters or shapes.[87,88]

When recording VA it is important to document the actual testing distance and M letters read, the chart used, and the use of any modifications (e.g., lighting levels). It is also important to observe the patient and record the use of eccentric fixation or adopted postures, together with any patterns of missed or incorrect letters.

Near visual acuity: Charts include single letter (reduced Snellen), unrelated word and continuous text charts. Single letter charts may be used for a quick estimate and for children or adults with limited literacy. Continuous text charts give a better understanding of performance in reading and reading related tasks. Continuous text charts include the MNREAD (available in many languages), the New Lighthouse, the Lighthouse chart for children, the Colenbrander charts (also available in many languages), the Radner charts, the Balsam Alabdulkader-Leat (BAL) charts in Arabic and the C-Read charts in Chinese. The MNREAD charts are also available on an iPad and have been shown to give equivalent results to the printed version.[89] All these will give a satisfactorily repeatable and valid measure of near VA in M print or logMAR for the calculation of magnification.

Continuous text reading cards allow measurement of maximum reading speed, critical print size and reading acuity. These measures are useful for determining prognosis for meaningful reading and for calculating the magnification levels required for various tasks. Reading acuity is often evaluated with each eye separately, as the eye which has previously been the patient's "better eye" may not always give the best reading fluency. Some patients may perform better for reading with one eye occluded, and this can be evaluated at this point in the assessment. Binocular reading performance should also be assessed, to determine any inter-ocular interference or enhancement. The SK Read Chart is designed on the logMAR principle and is composed of unrelated words and letters. It is designed to give additional information regarding the types of errors that are made due to central or paracentral field loss and can be used for training. The IResT charts are designed to measure reading speed for standardised paragraphs of text in a single print size, and are also available in multiple languages.

Lighting can have a substantial impact on both distance and near VA for people with low vision, so the effect of illumination on VA can be considered by increasing or decreasing the illuminance.[1] The optimal lighting level is dependent on the ocular disorder.[90,91]

3.1.3 REFRACTION

Objective and subjective refraction are fundamental components of a low vision assessment and spectacle correction should be optimized before pursuing additional devices. Often a significant VA improvement can be gained (11-16% of patients can gain a moderate to large improvement with refraction[44,92]) and it is important for the assessment and dispensing of most optical devices that the correct refraction be in place. Note that the pinhole is rarely useful to determine refractive change in patients with low vision because of the reduced illumination or presence of a central scotoma and need for eccentric fixation, and so an actual refraction is necessary.

Objective refraction with retinoscopy can be conducted as usual, although when the reflex is dim or less clear, alternative techniques should be explored. These include radical retinoscopy (closer working distance than usual),

bracketing neutral, off-axis retinoscopy, working in very low light levels, trialing high powered lenses and near (Mohindra) retinoscopy. Autorefractors can give an objective result when the media are relatively clear.

Subjective refraction is ideally undertaken in a trial frame because phoropter refraction may be limited due to factors such as the presence of central or peripheral scotomas and the need to make larger lens changes. Monocular refraction is typically employed, and non-standard distances can be used to allow the patient to see a line of letters. Correction to infinity can be applied afterwards.

Lens changes should be based on the concept of a just noticeable difference (JND), either determined by the guideline (denominator of the 20 foot Snellen fraction in feet/100) or by patient trial (starting with a large lens change, and reducing it based on the patient's ability to detect a change). For spherical refraction in presbyopes, the bracketing method is efficient. In pre-presbyopes, vision should first be fogged to relax accommodation by the addition of positive lenses, followed by progressive addition of minus (less plus). The highest plus (or lowest minus) lens of a pair of lenses which results in a "no difference" response is the correct sphere. Crossed-cyl technique can be used in both presbyopes and pre-presbyopes using higher powers of cross-cylinder lenses. Cycloplegic refraction is not contraindicated in most young low vision patients.

3.1.4 OCULAR ALIGNMENT AND MOTILITY

It is important to determine the ocular alignment of the patient, as it is relevant information for choosing binocular versus monocular devices. The Hirschberg test can be employed (although eccentric viewing may affect the apparent fixing eye). A cover test can also be employed with a suitably large target.

For patients with nystagmus, evaluation of the null point should be assessed during a motility test. This is best undertaken by pausing the target at different positions as the nystagmus may dampen with a stationery target.

3.1.5 CONTRAST SENSITIVITY (CS)

CS testing gives valuable information regarding the potential for reading and is predictive of difficulty with a wide range of other visual tasks (daily living skills, mobility, face discrimination, driving) and perceived disability.[14,15,93-101] CS may explain a patient's difficulty when VA is relatively preserved. Poor CS is also a risk factor for falls.[102,103] Often low vision patients have not previously had their contrast sensitivity measured. As a result, an important role for the low vision optometrist is to perform CS testing and to educate patients and their circle of care on the implications of any contrast sensitivity deficits.

Consequently, CS charts should be available to the LV clinician. Recommended and validated CS charts are the Pelli-Robson Contrast Sensitivity and Mars Letter Contrast Sensitivity charts. The measurements on these are interchangeable.[104] Other options are the Sloan Letter Low Contrast flip chart or the Rabin Contrast sensitivity chart. The Patti Pics or Lea symbols are available for young children. Low contrast acuity charts are available, but these do not give the same predictive information as CS charts, and have been less intensively studied. As the name implies, they give a visual acuity measurement for a target at a specific low level of contrast (e.g., 10%), and are thus not interchangeable with true CS charts in which the contrast changes to determine the threshold. Low contrast acuity charts may be useful for demonstrating to a patient the effects of contrast on their vision, i.e., how much their VA decreases with a reduction of contrast). There are mixed contrast charts for distance and near VA testing.

3.1.6 VISUAL FIELDS

Depending on the ocular diagnosis and the expected field loss, the LV clinician may measure central or peripheral fields.

Measurement of central visual field loss is the first step of eccentric viewing training. Central field loss can be estimated with the Amsler grid, although a negative result is not reliable.[105] Amsler grid at low light levels (e.g., with the NoIR 4% grey) can show field defects more reliably.[106] The tangent screen and its modifications (modified Amsler, California Central Visual Field test) can give useful information about central scotomas. The Humphrey can also be used, but threshold testing will be time consuming in LV patients. Microperimetry is the most accurate way of measuring central fields when there is a central scotoma.

Peripheral field loss can be documented with kinetic perimetry such as the Goldmann, or with static perimetry such as the Humphrey Field Analyser or the Octopus.

3.1.7 COLOUR VISION

Although colour vision testing is less critical than impairment of VA, CS or fields, many patients with visual impairment will have colour vision defects. Being able to measure colour vision and discuss it with patients is important. Blue-yellow defects are common in addition to red-green, so a test that can identify both types of defect is required. Pseudo-isochromatic plates may be of contrast that is too low and therefore maybe too sensitive for patients with low vision. A good test is the Farnsworth D15 (enlarged D15 for patients with lower levels of VA). Allowing the patient to bring the chips closer is acceptable.

3.1.8 GLARE TESTING

Many LV patients have glare difficulties. Although it is usually acceptable to rely on the patient's symptoms and undertake a tint assessment, glare testing may be useful in some cases (e.g., in documenting glare disability in a patient considered for cataract surgery). The Brightness Acuity Test is the clinical standard but an estimate can be obtained by measuring VA or CS in the presence of a glare source in proximity to the chart or with a transilluminator or penlight held close to the patient at an angle to his/her line of sight.

3.1.9 OCULAR HEALTH ASSESSMENT

Evaluating the ocular health of low vision patients is useful for relating structure to function of low vision patients. It allows clinicians to evaluate the state of the eye disease causing the visual impairment, and any progression from a previous assessment. For patients with more than one ocular disease contributing to visual impairment, examining the health and state of patients' eyes gives the clinician a better understanding of how each condition is affecting vision. The components of ocular health assessment may include:

- Anterior eye examination by slit-lamp biomicroscopy
- Tonometry
- Interior ocular examination by non-dilated fundus evaluation if possible

3.1.10 ADDITIONAL ASSESSMENTS

Ocular health assessment by dilated fundus examination

Dilated examination of the interior ocular structures is not routinely included in a LV assessment, as this is commonly undertaken in a separate, prior oculo-visual assessment or by the patient's primary care optometrist or ophthalmologist. However, there are occasions where the presenting symptoms, disabilities or measurements do not accord with the patient's diagnosis or when there is no recent ocular health assessment. In these circumstances, the LV clinician may have to postpone the low vision assessment in favour of an assessment of ocular health or arrange for a subsequent appointment for an ocular health assessment. The low vision assessment cannot proceed after dilation, as most measurements will be affected.

Imaging (e.g., Optical Coherence Tomography, ultrasound)

***Electrodiagnostic testing*:** ERG, VEP or EOG may be necessary to confirm or establish a diagnosis, rule out disease or obtain a measure of visual acuity in some cases.

3.1.11 CREATION OF THE INITIAL REHABILITATION PLAN

At the conclusion of the assessment, the LV optometrist is able to create an initial **Low Vision Rehabilitation Plan,** which may include any or all of the components described below. The plan should be disseminated in any reports that are made available so that all professionals are made aware of the findings and plan. Many components can be implemented by the low vision optometrist, together with an in-office low vision assistant or therapist. Implementation of the full plan may require referral to other service providers.

3.2 LOW VISION REHABILITATION – MANAGEMENT

The tools at the disposal of the LV clinician include optical, non-optical and electronic magnification, increased contrast, lighting control, minification, relocation of the object or image, training, and adaptations. Patient and family education and referrals to other service providers are also important components. As yet, there is little high level evidence that one specific type of evidence or approach is more effective than others (Appendix B) although, as described earlier, it is known that LVR as a whole is effective. Therefore, the fol-

lowing discussion relies on expert opinion and other research evidence that is available. Broad approaches are described, although ultimately what is effective for each patient should be recommended and prescribed.

3.2.1 MANAGEMENT OF CENTRAL VISION LOSS

The majority of patients presenting in low vision settings have some level of visual acuity reduction and will benefit from magnification for near and distance tasks. The most frequent goal of patients is reading and other detailed tasks such as sewing, writing, and watching TV.

Near Magnification

Magnification requirements may differ depending on the task required by the patient. There are several ways to estimate starting magnification for near, and these are reviewed in Appendix D. Most of these methods take into account the patient's task requirements in terms of a target print size. Note that these estimates are starting points for the magnification or equivalent viewing power (EVP)/near add required and that higher, and sometimes lower, magnifications should be trialed.

Optical devices for near magnification

Spectacle mounted reading lenses/high adds/microscopes: These devices are spectacle-mounted plus lenses consisting of a near addition (plus power) to allow close focus to produce relative distance magnification. Some examples include single vision readers, bifocal near additions, prism half-eyes or clip-on lens. The power is determined as described in Appendix D. These can be demonstrated in a trial frame, with ready-made prism half-eyes or microscopes. Custom reading glasses or microscopes address the needs of those with significant anisometropia or astigmatism, and may provide superior optical quality. Head-band, clip-on and bar-mounted microscopes are available for tasks which require a greater working distance, which is obtained because the lens to eye distance is increased.

In an absolute presbyope, the patient's working distance in using the device should be the focal length of the near addition. If the patient has accommodation, it would supplement the power of the add. The working distance would then be closer than the focal length of the device. The method for estimating the reading addition in pre-presbyopes is described in Appendix D. Microscopes can be prescribed binocularly up to a +12D, or even 14D, add, but decentration and base in prisms must be considered with higher adds due to the near working distance and demand on convergence (see Appendix E).

Hand magnifiers (HM): These devices comprise a plus lens mounted on a handle that the patient holds at a distance away from the object. They are often prescribed for brief reading tasks and can range in magnification, size and illumination. A certain amount of dexterity is required of the patient as the distance between the magnifier and object must be maintained to view properly.

HMs can be trialed with power equal to the EVP calculated for a microscope. When the patient views through the distance portion of the spectacles while using the magnifier, the EVP obtained equals the power of the hand magnifier irrespective of distance, and the print or object being viewed should be held at the focal length of the magnifier. However, when the patient views through their reading addition while using the magnifier, the total EVP is dependent on the distance that the HM is held. The EVP obtained is less than the power of the magnifier when the HM is held further than the focal length of the hand magnifier. Thus, when the patient holds the HM further than its focal length, the patient should view through the distance portion of spectacles to gain maximum EVP. When it is held closer than the focal length of the HM lens, the patient can either view through the distance or reading portion of their spectacles. In all cases, the field of view increases as the distance between the magnifier and eye decreases.

Stand Magnifiers (SM): A stand magnifier is comprised of a positive lens mounted in a stand that sits on the object (usually the page). Dome magnifiers are classified as stand magnifiers – the thickness of the glass acts as the "stand". Stand magnifiers may benefit patients with dexterity issues, as the distance between the magnifier and object is fixed. They can be advantageous compared to hand magnifiers when a higher lens power is required and when steadiness and lens positioning become more critical. They are available with or without internal illumination. The preliminary testing with different light levels will indicate which type is likely to be beneficial. The light exiting a stand magnifier is divergent to a greater or lesser extent, creating a virtual image that is closer than infinity. This means that either accommodation or a reading addition is required to obtain a clear image, and the low vision op-

tometrist needs to be aware of the emergent vergence (image position) for each stand magnifier in his/her inventory (Appendix F). Stand magnifiers can be trialed with a transverse magnification (TM, sometimes called enlargement ratio) equal to the estimated magnification (Appendix D). The transverse magnification is not equal to the magnification marked on the device (nominal or trade magnification), but it is often available in the manufacturer's technical specifications and can also be calculated in advance by the LV optometrist. Similarly, the emergent vergence can be measured or found in suppliers' or manufacturers' catalogues or technical specifications.

Telemicroscopes: A telemicroscope is an afocal telescope focused for close work by a positive lens placed on the objective lens. These can give magnification for either near or intermediate tasks. They tend to be used for more specialized hands-free tasks, which require longer working distances. They can be prescribed in terms of magnification (if the patient's acuity is measured at the task working distance) or equivalent viewing power. Formulae are provided in Appendix F.

Often an example of each type of device will be trialed. The final type of device should be considered in terms of the patient's preference and ease of use, binocular versus monocular performance, hands free needs and lighting requirements. The magnification or EVP should be increased (and in some cases decreased) to obtain the optimum performance in terms of reading fluency or task performance, endurance, and acuity through the device. Higher magnification may provide better fluency at the patient's goal print size,[1] while lower magnification may be sufficient and be easier to use.

Contact lenses: Contact lenses can be considered for high myopes, as they will lose some spectacle minification. Possible undercorrection with a spectacle lens over-correction may be beneficial, so that the patient can still benefit from the relative distance magnification gained by removing their spectacles.

When optical magnification is insufficient for the task

Reading (and writing) are complex tasks. There are occasions when a patient is unable to read their goal print fluently despite trialing a range of devices and magnifications. The most common causes for this are:

- a very large and dense central scotoma (requiring a preferred retinal locus [PRL]) that is far from the anatomical fovea
- very poor visual acuity, such that a sufficient acuity reserve cannot be obtained
- poor contrast sensitivity
- restricted visual fields, such that there are insufficient characters within the visual field
- paracentral scotomas located in positions within the visual field that are critical for reading.

There are indications that an acuity reserve less than 2x, contrast sensitivity which is <1.00, a PRL which is >15° away from the anatomical fovea or a field of view that is <5 characters across will severely restrict reading rates.[1,94-96] In these cases, electronic magnification, eccentric viewing training, and/or non-optical solutions should be explored. However, optical magnification may still be useful for certain brief reading tasks.

Electronic magnification and accessibility options: Electronic magnification and accessibility options should also be considered for many patients. This may include a discussion of the accessibility options which are now available on current devices (mobile phone, tablet, laptop). Accessible features include text to speech, voice assist, talk-back and magnification, contrast, font and colour options. Adapted computers (software and hardware adaptations) can be demonstrated, or an assessment can be recommended.

A demonstration or assessment for video magnification, such as hand-held, portable and desktop video magnifiers (CCTVs), is often indicated. Video-magnification is particularly useful when a patient has poor contrast sensitivity (because of the contrast enhancement options). Portable video magnifiers may be a useful supplemental device for patients who also have optical devices and perform well with them. They can be considered for similar tasks as a stand magnifier. Optical aids may be used more frequently and for more tasks, while hand-held video-magnifiers may allow reading of smaller print and be preferred for leisure reading.[107,108] Because of their variable magnifica-

tion, hand-held video-magnifiers may be a cost effective solution.[109] For more prolonged tasks, the functionality of a portable video magnifier should be compared against a desk-top video magnifier. Desk-top video magnifiers are also useful when patients require higher levels of magnification, variable magnification due to disease progression, or assistance for writing and extended reading, or have constricted or hemianopic visual fields. Reading speeds may be higher with the use of CCTV devices compared to optical magnification even with eccentric viewing training.[110]

Writing

People with central vision loss may also require magnification for writing. Typically, less magnification is required for writing compared to reading. Often, a target of 2M may suffice, which frequently means about half the magnification compared to reading. Thus spectacle-mounted devices may be an option for writing even if they are not suitable for reading. There are optical and electronic stand magnifiers with a gap in the stand on one side, which allow for the use of a pen. Clip-on, bar mounted, or head-band magnifiers may also be options for this activity. Video magnifiers (desk-top types and hand-held or portable CCTVs mounted in a stand) and adapted computers may be considered as writing devices.

Distance magnification

Although the management methods for distance are more limited in scope than the variety provided for near work, there are still a number that can be applied to increase the quality of life. Telescopes are the main method of providing magnification for distance tasks (although in some cases relative distance magnification can be achieved, e.g., decreasing the viewing distance for TV).

Optical devices for distance magnification

Telescopic magnifiers: Telescopic magnifiers can be categorised as Galilean or Keplerian, handheld versus spectacle mounted, monocular versus binocular, and ready-made or custom. The choice is based on the magnification required, and the goals and abilities of the patient.

Galilean telescopes have the advantage of being lighter and less expensive than Keplerian telescopes, but are limited to ≤4x magnification. Therefore, they serve best for patients with minimum magnification requirements. Keplerian telescopes, sometimes called prism telescopes, have a large range of magnification (up to 8x, or at most 10x), but due to their multi-lens system, they tend to be bigger, heavier and more expensive than Galilean telescopes.

The decision regarding a telescope depends on task demands (e.g., hands-free, duration), field of view, magnification, binocularity, and the patient's refractive error (whether this needs to be incorporated or whether the telescope can be focussed to compensate). While spotting tasks (e.g., reading signs, checking cross-walk lights) can be aided by handheld telescopes, spectacle mounted telescopes should be considered for extended tasks (e.g., watching TV, live spectator events) to prevent fatigue and improve stability. Spectacle-mounted options include those positioned in the primary position, upper bioptic or, for a telemicroscope, the lower bioptic position. Clip-on telescopes are also available. In some countries, bioptic telescopes can be used for driving. This is not generally accepted in Canada at present, but may be allowed on a case by case basis in some provinces.[111]

Head-mounted Video Magnifiers: Head-mounted video magnifiers are developing rapidly and becoming more widely available. These can be considered for patients who have multiple goals. These devices are typically autofocus and provide variable magnification at a range of distances plus various contrast and digital enhancement options. As a result, electronic magnifiers have been able to augment vision in more ways than a typical telescope. In one report, despite a learning curve in handling the device, visual function improvements were gained rapidly and were sustained over three months of use. Self-reported function continued to improve over that time.[112] Therefore, the visual acuity obtained on the initial trial visit tends to be a good estimate of the overall improvement that the patient can expect from utilizing the device. However, it should be stated clearly to the patient that the current versions of these devices cannot be used for driving or mobility. These electronic devices tend to be of a higher cost than optical magnifiers. There is, however, a recent trend to harness the power of existing smartphones to perform similar functions as dedicated head-mounted devices but at a lower cost.[113]

Low vision optometrists should be aware of products which function with integrated cameras along with user input (e.g., pointing) in order to identify an object or read a passage to the patient. This information may be provided to the patient either via audio feedback or direct conduction of sound via bone.[114,115]

For any magnification device, different potential devices should be demonstrated to the patient for each task, allowing the patient to select the optimum device for their needs based on the following considerations:

- Specific task to be accomplished (e.g., requirement for hands-free)
- Duration of the task (long-term viewing versus spot-checking)
- Cosmesis (spectacle mounted versus separate device)
- Weight
- Contrast and brightness
- Cost
- Ease of use

In-office training visits followed by a take home trial are helpful, and follow-up phone calls after a month or two should be used to check for problems, difficulties with devices or further rehabilitation needs. The feasibility of telerehabilitation for low vision (training and evaluation of the patient through videoconferencing) has been considered,[116] although there are no randomised controlled trials of the effectiveness of this approach[117] (see Appendix B).

Eccentric viewing

Eccentric viewing is an adaptive trait that naturally develops in patients who have a dense central scotoma in the better-seeing eye. It involves the use of a preferred retinal locus (PRL) or non-foveal area which is outside the patient's scotoma area and is used for fixation. The purpose of eccentric viewing training is to aid the patient to develop a more consistent and efficient use of their natural PRL, to improve fixation stability at the PRL, to hasten the development of a PRL and/or to optimise its position.[110,118] There is, of course, variability in the optimum placement of the PRL based on the size, shape and position of the scotoma. Most patients naturally place the scotoma in the superior or, unexpectedly, in the right visual field. Placement in the right visual field is not expected to be optimal for reading (when reading left-to-right). In people with normal vision, reading with a simulated scotoma is more successful using the inferior, rather than superior, visual field[119], and with the scotoma moved to the left, rather than right.[120] However, for people with AMD, studies have shown that reading rate is not strongly related to the position of the PRL,[121,122] although Watson et al. show that reading errors were more frequent when the scotoma was positioned above or to the right.[123] The clinical goal is to develop a PRL such that there is the maximum horizontal area of intact visual field extending to the right, and which is closest to the non-functioning fovea (for best VA). In the case of a symmetric, well-centred scotoma, the preference is often to move the scotoma upwards in the visual field.[110]

The usual components of eccentric viewing training are briefly listed in Appendix G. See Leat et al.[110] for a more detailed description:

Neither the effectiveness of eccentric viewing training nor the effectiveness of one method of eccentric viewing training over another has been well-established, as there are few well controlled, high quality studies (see Appendix B).[110,118,124] Many studies did not have control groups, were not masked, included other interventions concurrently with eccentric viewing training, or did not have long-term follow-up. There is some evidence that eccentric viewing training may improve near VA[110] and vision for general tasks such as shopping or household chores.[125] Some studies have shown slight improvements in reading speed with eccentric viewing training,[126-128] while Seiple et al. demonstrated some increases in reading speed with saccade training.[129] However, Hassan et al. did not find increased fixation stability, although the position of the PRL did change after traditional eccentric viewing training.[130]

Recently there has been increased interest in the use of biofeedback.[131] Microperimetry can give a more precise measure of the central scotoma and may also incorporate biofeedback.[126,132,133] Studies of these methods have been mostly longitudinal cohort studies without randomisation or masking. The results suggest some potential for these methods, but they have not been compared against traditional eccentric viewing training and can only be implemented monocularly.

The time and effort spent on eccentric viewing training may depend on the funding available. Leat et al. showed that the provision of a desk mounted video magnifier resulted in significantly faster reading than eccentric viewing

training for patients with age-related macular degeneration.[110] However, eccentric viewing training still retains a place in the range of approaches available for LVR.

A more controversial approach is yoked prism relocation to enable the patient to orientate towards, and use, their PRL. The concept behind this method is to aid steady fixation by reducing the patient's need to move his/her eyes to use their PRL. However, little consistent improvement has been demonstrated and it is not a recommended treatment at this time.[134] The lack of effect may be due to refixation behind the prism or lack of accuracy in properly determining the PRL, and the purpose of this approach is unclear when a PRL already exists. In fact, one well-controlled randomised clinical trial of the long term effect of such prisms not only found no benefit, but reported harmful effects such as dizziness, headaches, and strain.[135] Considering the increased risk of falls in low vision patients, especially older patients, yoked prism relocations strategies should be avoided.

3.2.2 MANAGEMENT OF CONTRAST SENSITIVITY LOSS

Reduced contrast sensitivity is the other main category of vision impairment causing disability alongside central vision impairment (VA loss) and visual field impairment. It is an additional and important measure of visual function, as poor CS may exist when VA and visual fields are fairly intact.[136] CS of <1.6 on the Pelli-Robson chart represents a visual impairment (i.e., outside the normal range).[104,137] When CS is reduced to <1.40,[14,15] the patient is likely to be experiencing some disabilities such as issues with mobility and resolution tasks, but when CS is <1.00 visual performance is severely compromised, even with appropriate magnification. For example, reading is likely to be slow even with the use of optical magnifiers.[94-96]

Managing contrast sensitivity loss is difficult. There are three approaches; 1. change the task parameters so as to optimise the patient's contrast sensitivity, 2. increase the contrast of the task, 3. use vision substitution methods.

Changing the patient's contrast sensitivity

Some patient's contrast sensitivity may be improved at lower or, more frequently, higher levels of illumination. This is one explanation for why lighting levels are critical for low vision patients. Note that increasing the illumination on print does not change the contrast of the print, but changes the contrast sensitivity of the patient. Reducing the light scatter within the eye may result in a more contrasted retinal image for some patients. This may be achieved with the use of a typoscope or electronic reverse contrast for print (white on black), which allow high illumination of the print without increasing veiling glare due to intraocular light scatter. It has been suggested that shortwave length absorbing filters also reduce light scatter within the eye, thereby enhancing the contrast of the retinal image. However, although patients do report benefit, studies show very little or no objective improvement on visual functions such as VA, contrast sensitivity, or reading with coloured filters[108,138,139] (see Appendix B).

Increasing the contrast of the task

Contrast enhancement options exist in all modern video magnification devices so that poor contrast print, such as newspaper, can be electronically displayed at almost 100% contrast. Contrast of photos and writing can be similarly increased. There are also contrast accessibility options on computers. In some head-borne electro-optical devices there are digital enhancement options such as edge enhancement, which may also help to increase the visibility of pictures, faces, TV or features in the environment.

Environment modifications, such as marking the edges of steps, stairs and doors can be considered. Techniques for increasing the contrast when eating and cooking include using plates or cups of a contrasting plain colour. For writing, use of black felt tip or marker pens, and bold-lined paper can be helpful.

Vision Substitution

When CS is very poor, sight substitution methods should be considered for speed of access to information and ease of undertaking tasks. This can be supplemental to visual access, i.e., patients do not need to commit solely to one or other strategy. Voice command and output exist on most smart technology and laptops, or specific voice output software such as Jaws, can be installed. Dedicated, stand-alone optical character recognition (OCR) scanners are available for individuals who do not use computers. Players and readers (e.g. Daisy player) may be appreciated by patients who want access to auditory books. Other options include devices such as watches, calculators and liquid level indicators with auditory output.

3.2.3 MANAGEMENT OF PERIPHERAL FIELD LOSS

Conditions such as retinitis pigmentosa, choroideremia, glaucoma and cerebrovascular accidents can constrict or reduce the available visual field. Patients with less than 70° total solid angle of visual field are likely to experience

mobility difficulties.[12] Other difficulties that patients with peripheral field loss will encounter include more difficulty with daily living skills, dark adaptation and locating objects, resulting in an overall reduced quality of life. Patients can be helped with certain devices, techniques and training.

Options for general peripheral awareness

Prisms: For patients with homonymous hemianopia, Peli prisms or sector (spotting) prisms can be demonstrated and discussed.[140,141] There are a variety of methods for placement of prisms.[142] Note that sector prisms are often described as being placed on the spectacle lens on the side of the field defect, but this leads to diplopia when the patient looks through the prism. An alternative is to place a sector prism on both spectacles lenses, as shown in Appendix H,[143] but this still results in an apical scotoma. Peli prisms increase the visual field by creating areas of visual confusion. For a detailed discussion see Apfelbaum et al. who compared the optics of sector prisms and Peli prisms.[144]

Patients with overall peripheral constriction may benefit from sector prisms on both sides of the spectacles and in the lower visual field, or a channel lens.

A useful method for placing sector prisms is to slowly introduce a sticky paper, such as a Post-It Note, from the side of the visual field loss while the patient looks straight ahead. Mark the point where the patient first sees the paper, and then place the prism 1-2 mms temporally to this mark. The patient should not be aware of the edge of the prism when viewing in primary gaze.[142,143] Training is a vital component for the patient's successful use of any of these prisms.

Minifiers: For patients with advanced contraction of the visual fields, minifiers, such as reverse telescopes and handheld minus lenses, can be considered. Reverse telescopes can be spectacle mounted in a bioptic position or hand held. Amorphic lenses (which have minification in the horizontal meridian only) have been available in the past.

Training: Visual search training is important in cases of significant field loss (hemianopia, constricted fields) to aid the patient to learn deliberate eye movement strategies to scan their environment. Referral for orientation and mobility training, sighted guide techniques and guide dogs are options to be considered.

Options for reading for patients with field loss

Once patients with conditions such as RP and glaucoma have significantly constricted visual fields, central vision is also often affected with both contrast sensitivity and VA being reduced. In this situation the patient may require magnification, but there is an additional limitation due to the reduced field. Too much magnification may result in poorer reading ability, if insufficient numbers of characters can be viewed at a time. In these cases, calculating magnification from the CPS may be effective. Video-magnification aids and computer software adaptations may be effective for patients with either constricted fields or hemianopia by allowing scrolling of the text through the remaining visual field, reducing the column width of the text and, for those with reduced CS, increasing contrast.

Patients with hemianopia often have difficulty tracking the line of text and tend to omit the endings or the beginnings of lines. Strategies such as marking the text margins, using line guides, and holding the print vertically or diagonally may help.

3.2.4 MANAGEMENT OF NYSTAGMUS

Positioning of visual material is a useful strategy for patients with nystagmus. Reading material can be positioned at the null point of nystagmus and the patient can position themselves so that the null point can be used more comfortably, without having to turn the head. For example, if the null point is to the patient's right, then visual material should be placed to their right and they would be more comfortable sitting to the left of the class or theater so their gaze is directed to the right. Yoked prisms may be used to move the null point closer to the primary position. In cases where convergence reduces the amplitude of nystagmus, base out prisms, with the incorporation of additional minus for pre-presbyopes, can be considered. Contact lenses may have benefits for patients with nystagmus, by decreasing the amplitude and possibly improving visual acuity and contrast sensitivity slightly, although not all studies have demonstrated improvements.[145,146]

3.2.5 LIGHTING REQUIREMENTS

Many patients with low vision have a limited range of light levels for optimal performance (either higher or lower light levels may be optimal) and experience more glare.

Lighting
Lighting can be considered simultaneously with magnification, as increases in lighting may frequently change the requirement for magnification (often decreasing it). Lighting of different colour temperatures and different intensities can be demonstrated. Goose neck lamps which include LEDs of different colour temperatures are readily available and illuminance can most easily be changed by varying the distance between the page and the light source. The relationship between illumination and the distance of the light source from the page is not linear, however, but behaves according to the inverse square law. This states that the illumination is proportional to the reciprocal of the square of the light source output. In other words, bringing the light source 3x closer results in 9x more light on the page. This can be explained in simple terms and demonstrated to the patient, so that the patient is simultaneously educated about their lighting requirements. If an illuminated HM or SM is the device of choice, then the lighting is inbuilt. If this is not the case, the lighting can be specified for the patient after measuring the illuminance of the preferred lighting with a light meter (measured in Lux). This can be transposed to the lumens required for a light source at a known distance according to Appendix I. The patient can be advised about the light source that they require in terms of colour temperature and luminous output (Lumens).

The positioning of lighting is also important. Usually for close detail tasks arrange the light to be over the shoulder, so that good lighting, but not glare, is introduced. The concept of task lighting is important, i.e., that specific and adequate lighting should be positioned in the areas of the home where tasks are performed.

Tints and filters
Non-selective and selective transmission tints can be of great benefit to many patients with low vision to control light levels and glare and to optimise patient comfort. A range of filters, such as short wave-length absorbing lenses, should be available for demonstration and are available from several suppliers. Other filters, which are often selected by patients, include blue, plum, grey and polarising filters. Conventional spectacle tints in grey or grey-brown can be useful for some patients, and can be prescribed in a photochromic lens. However, these conventional spectacle tints may not be sufficient for patients who are very photophobic.

Filters are currently assessed subjectively, either indoors or outdoors or both, depending on the patient's symptoms and the situation in which they experience most glare. Generally, non-selective transmission tints should be prescribed, unless the patient specifically benefits from a selective tint, as selective tints will always distort colour perception. Although short wave-length yellow tints may be subjectively beneficial to patients with glare, currently there is no evidence that they improve contrast for reading for patients with poor contrast sensitivity or that they improve reading speed. In fact, reading speed may be decreased (see Appendix B).[108] For young children, observation of the extent to which they open their eyes, relax their forehead, or lift up their head can be used to assess the optimum tint.

Filters can be prescribed in non-prescription glasses, prescription spectacles, clip-ons or fit-overs, depending on the patient's refraction and preference. For people who are very glare sensitive and photophobic, wraparound glasses or side shields should be considered. Patients with albinism or aniridia may benefit from an iris imprint contact lens with an incorporated tint, while those with achromatopsia may find a red or red-brown contact lens with a tinted pupillary zone very helpful.

3.2.6 OTHER INTERVENTIONS AND CONSIDERATIONS

Visual Training and Adaptive Strategies
Training should take place for all devices that are prescribed or recommended. This should include how to use and clean the device and how to replace any batteries. For reading, how to scroll the print across the field of view is important for higher magnifications. Training is typically delegated to an in-house, trained optometric assistant or low vision therapist.

Driving
Patients with central vision loss or peripheral loss may fail to meet the driving standard in their province of residence. In some provinces, and most states of the US, patients can continue to drive with bioptics. In the case of visual field loss, most provinces will consider driving on a case-by-case basis. For a summary of driving requirements, and ability to use bioptics according to province, see the AAA website.[111] The LV optometrist should be aware, however, that there is no clear association between moderate VA impairment, poor stereoacuity, or dependence on monocular vision and driving ability. Contrast sensitivity impairment is more consistently found to be associated

with driving ability, while driving with visual field loss shows a large variability between individuals; some people seem able to compensate with scanning eye movements, while others are less able.[26,147]

Non-Optical Options
The LV optometrist should be able to recommend the following:

- Devices which give relative size magnification, i.e., increase the size of the task, such as large print books, clocks, watches
- Devices with voice or auditory output, e.g., talking clocks, blood glucose monitoring for diabetics, audio books, liquid level indicators
- Access to auditory information, e.g., talking books, auditory newscasts
- Tactile or visual markings for appliances
- Reading guides, writing guide and signing guides
- Black pens for writing
- Environmental modifications including lighting, use of contrast and de-cluttering should be discussed. Literature or websites which illustrate these modifications are helpful.

In each case, the optometrist or his/her low vision assistant should be able to supply the devices or inform the patient where they are available. The LV optometrist may develop their own printed literature or have available leaflets from various other services which illustrate these approaches.

Additional Services
The LV optometrist must be mindful of other services that may be required by the patient and refer or recommend these when indicated. For patients who require Level 3 LV (Comprehensive LVR) inter-disciplinary care is indicated. This may be provided in one location as in a multidisciplinary clinic, or by close communication between service providers. When referring, it is recommended that the optometrist include their rehabilitation plan, including what interventions that have been explored and implemented.

Additional services include:

- Orientation and mobility training
- Occupational therapy, independent living specialist assessment
- Low vision therapy
- High technology assessments
- Social and community services
- Counselling
- Genetic counseling
- Surgical consultation, e.g., for cataract, nystagmus, strabismus
- Vocational counselling

3.2.7 NON-VISUAL FACTORS AFFECTING THE LVR OUTCOME

Psychosocial

The patient may be coping with the stages of grieving[148] and may not have reached the stage of acceptance, when providing low vision devices is usually more successful. This may limit the success of LVR. However, the LV optometrist should still demonstrate some interventions which, if accepted, may alleviate a patient's vision specific distress[33,149] and allow for the introduction of other options in the future. The optometrist should also arrange to see the patient for follow-up appointments and suggest counselling.

The optometrist must be sensitive to the link between visual impairment and poor mental health (depression, anxiety, feelings of isolation and poor self-esteem) which can lead to increased mortality[33,38] and be prepared to refer the patient for other services, such as counselling, when these are suspected. This need may be indicated by the PHQ-2 screener used at intake.

Cognitive factors

People with visual impairment are more likely to have cognitive losses.[150] Specifically, better near visual acuity and having spectacles for near work seem more highly associated with higher cognitive function than the level of distance VA.[151-153] Significantly, there is evidence that visual impairment precedes cognitive loss or dementia (i.e., is a likely causative factor) and this remains true when adjusting for other factors, such as educational level, income or hearing loss.[151,154-158] Also, in several studies, vision loss was more predictive of cognitive decline than hearing loss.[153,157,159]

Therefore, treatment for vision loss, including refractive correction or visual rehabilitation, may help to prevent cognitive loss or improve cognition. There is evidence that treatment of cataract[160-162] or other eye disease[154] helps to improve cognitive function or prevent cognitive decline. There are fewer studies regarding LVR. Meyniel et al. documented that patients with cognitive impairment who underwent four months of LVR improved in their average cognitive function.[163] Zheng et al. concluded that maintaining good vision, in particular near vision, may help protect against cognitive decline in older years.[158] One proposed mechanism for the prevention of cognitive decline is the ability to continue cognitively stimulating activities, such as reading, and maintain social networking.[154,158] If this mechanism proves correct, then visual rehabilitation should not only enable patients to perform desired tasks and improve quality of life, but also enable them to maintain cognitive function.

Lastly, a different issue relating to cognitive impairment and LVR is the extent to which patients with cognitive impairment may benefit from LVR. There are some cohort studies which address this question. Hagerman et al. reported that patients with cognitive impairment attained improvements in VA with devices.[164] Whitson et al. trialed an enhanced low vision rehabilitation programme in a small cohort of people with cognitive deficits and demonstrated improvements in vision and cognitive function. Patients were able to benefit from training with a CCTV.[165] Gervais et al. in Quebec described a case series of patients and concluded that cognitive deficits do not preclude successful LVR.[166]

Co-morbidities

Other co-morbidities will impact the effectiveness of the LVR plan. For example, patients with diabetes may have more difficulty adapting to vision loss, while those with paresis due to stroke or arthritis will have physical difficulty managing hand-held low vision devices. Post-stroke patients experience a wide range of both visual and systemic difficulties in addition to hemianopia. These include paresis, perceptual changes (such as neglect and midline shift), incommitancies with variable diplopia, loss of visual acuity and aphasia. These need to be addressed and taken into consideration in the rehabilitation plan and may require referral for optometric neurorehabilitation, which is outside the scope of this Guideline.

Patients with developmental delays represent a population with unique needs. In this population, the emphasis is to first undertake a good visual function assessment (description of visual abilities). From this evaluation, recommendations regarding optimum lighting, size, contrast, crowding (or lack of) and positioning of visual information, use of good colour contrast, vision stimulation, vision therapy, and refractive correction can be made.[167]

Age

LVR should be considered from the moment that a child is diagnosed with a condition which results in a visual impairment. Such a diagnosis in a child has been described as a rehabilitation emergency and there are specific approaches for young infants, which are beyond the scope of this document. In fact, this is described as habilitation (rather than

rehabilitation) as the child has not lost skills, but rather needs help to develop skills in the first place.[168,169] For an approach to Pediatric Low Vision Rehabilitation see Leat (2015).[168]

LVR should not preclude older adults either. Jackson et al. found that female patients aged 85 years and older gained as much benefit from LVR in overall ability and reading ability as younger patients (though not in mobility).[170]

4. CONCLUSION

LVR is an on-going process for most patients and follow-up is important. Vision may alter and new devices become available. Patients' acceptance level, activities and goals may change over time. Patients describe it as a journey. Most patients, especially those with poorer vision, will require multiple devices and strategies for different tasks, and these may be added over time.

To conclude, optometrists are uniquely qualified to provide LVR as they can undertake refraction, optimise visual function with spectacles and contact lenses, accurately assess visual function and understand the impact of ocular conditions, develop a vision rehabilitation plan, prescribe optical and non-optical, hand held and spectacle mounted devices, provide vision training, advise about visual strategies and environmental modifications, and co-ordinate with other services. LVR requires a holistic approach to the patient, and the LV optometrist must be mindful of the emotional and psychological state of the patient. Lastly, interventions that are recommended should not only be task(s) specific, but also patient specific, i.e., tailored for each particular patient's goals, requirements and limitations.

Acknowledgements

We would like to thank Roanne Flom and Nicole Ross for reviewing this document and providing detailed feedback. We also thank Douglas Brunton and Kristin Snell for proof reading the document.

Appendix A

Provincial Health funding for Low Vision Assessments and Devices

Courtesy of, and adapted from: Shamrozé Khan and Susan Leat

Note that lack of funding does not preclude optometrists from providing LV assessment and devices privately. Funding is listed when known. BCVA = Best Corrected Visual Acuity

Province/ Territory	LV Assessment by Optometrist	Low Vision Devices
Canada-wide	Funding up to a maximum exists for some veterans.	Funding up to a maximum exists for some veterans. They may be required to access provincial funding first.
Canada-wide	There may be coverage for seniors, those on income support programmes, employees through the employer, children, and through 3rd party insurance.	There may be coverage for seniors, those on income support programmes, employees through the employer, children, and through 3rd party insurance. Devices that are medically necessary may be covered through special requests.[171]
PEI	None	None
Nova Scotia	Yes, low vision assessment once every 2 years can be billed at 30 Medical Service Units (MSU) with one mandatory follow up (15 MSU). Acuity must be at least 20/50 or worse in the better eye.	None
Newfoundland and Labrador	None	None
New Brunswick	None	None
Quebec	Restricted. There is a fee for an optometrist ($70) doing a LV assessment in their office for people who meet the QC definition of visual impairment (as below) and <19 or >65 years old. Covered for all ages if seen in Government funded Multi-disciplinary Rehabilitation Centers. There are 18 of these centers across the province.[172] The eligibility criteria and the list of visual aids can be found on the RAMQ website.[173] Patients who meet the visual criteria can be seen by the optometrist and all the other professionals (OT, psychologist, mobility instructor, etc...) for free in these centers if they have a valid RAMQ card. Eligibility criteria are a BCVA of less than 6/21 in each eye or a visual field of less than 60 in the horizontal and vertical meridians or complete hemianopia or a VA of 6/18 or less "for persons who suffer from a degenerative visual problem, visual impairment, physical deficiency (motor, hearing or speech), or an intellectual disability".	LV devices (long cane, magnifier, CCTV, etc...) are covered and provided on on-going loan only when the patient is evaluated through one of the government-sponsored rehabilitation centres and when they meet specific criteria which depend on the device (e.g., CCTV are covered for patients whose VA is 6/60 or less) and based upon the patient's need (e.g., require it for study or work or who live alone).
Ontario	None	Assistive Devices Program (ADP) provides partial coverage (up to 75%, 100% for those on income supports) for both low and high tech devices. Available for a person who "is unable to perform common everyday, age-related visual tasks due to reduced visual functioning level" Devices must be prescribed by Optometrists registered with ADP. There is a lease programme for CCTVs.[174]

Province/ Territory	LV Assessment by Optometrist	Low Vision Devices
Manitoba	Partial. Per diem available for services provided through VLRC, but not when provided without VLRC.	
Alberta	Partial. For ages <19 and >64, B660 "Examination for low vision aid" can be billed if the optometrist has specific equipment and a certain number of LV devices available, and can demonstrate or refer for non-optical aids, electronic aids and O and M assessment.	The CNIB Specialized Technical Equipment Program (STEP)[175] is a government-funded subsidy program administered through VLRC service centres.[176] Coverage for up to 75% of device cost if patient qualifies (VA is 20/200 or poorer or the visual field is severely restricted and based on financial need) and up to 100% if low income. But only 40% of those that apply receive the funding. This includes high-tech aids such as CCTVs, Zoomtext, computer software, OCR. Those with a BCVA of less than 20/70 or with severely restricted visual fields, can qualify for assistance for low-tech aids.
Saskatchewan	Restricted. Only covered if provided at the Low Vision Clinic at the Pasqua Hospital upon referral by an optometrist or ophthalmologist.	Coverage and subsidies for some devices through the Saskatchewan Aids to Independent Living (SAIL) programme, which is operated though CNIB/Vision Loss Rehabilitation Saskatchewan.[69,177] Eligibility depends on the device and the level of VA. For example, conventional hand-held and stand illuminated magnifiers are funded at VA of 20/70 and poorer; various high technology devices at VA of 20/150 or worse or fields of less than 20 degrees; digital portable video magnifiers and iPad at 20/200 and poorer. Telescopes/ spectacle mounted telescopes are only covered though Labour Market Services. Low vision clinic services are available for children with any level of reduced acuity. Tints are covered for children through SAID but require an OD report requesting this because of medical necessity. =
British Columbia	Partial. The MSP code 2892 is billable every 6 months if the optometrist has the appropriate equipment. There are other MSP fees that may be billed in conjunction with the 2892 code. The optometrists must obtain prior approval to bill these codes. Balance billing is allowed.	None
Nunavut		
Northwest Territories	Partial. On application, Government funds eye clinic and travel to communities.	
Yukon	None	

Appendix B

Summary of Systematic Reviews of Low Vision Interventions

RCT = Randomised Controlled Trial; QoL = Quality of Life; LVS = Low Vision Services; AMD = Age-related Macular Degeneration; PRL= Preferred Retinal Locus; O&M = Orientation and Mobility; ADL = Activities of Daily Living; IADL = Instrumental Activities of Daily Living

Authors, Year, title	Main question	Inclusion criteria	Search results	Main conclusions	Comments
Barker et al., 2015[178]	The effectiveness of optical aids compared to standard optical refractive correction in children and young people with low vision	RCTs or quasi RCTs, including within person designs, children and young people aged 5-16 years	No studies met the inclusion criteria	There is a lack of high quality evidence of the effectiveness of optical aids for this age group.	Very strict inclusion criteria, so lower levels of evidence not assessed
Binns et al., 2012[46]	The effectiveness of different models of LVS provision	Not clearly stated	58 studies met the liberal inclusion criteria of which 7 were RCTs	There is sufficient evidence to confirm that low vision rehabilitation improves clinical and functional outcomes. Despite different models of LV care, most studies showed improvement in functional ability. Less clear evidence on QoL outcomes.	The authors were unable to conclude whether one model of LV service is better than another.
Bittner et al., 2015[117]	To compare the effects of telerehabilitation with face-to-face (e.g., in-office or inpatient) vision rehabilitation services for improving vision-related QoL and reading speed in people with visual function loss	RCTs, Patients with any cause of vision loss	No studies met the inclusion criteria	There is a need for clinical trials to explore this mode of delivery	
Gaffney et al., 2014[118]	The effectiveness of eccentric viewing and steady eye strategy training in people with central vision loss	Participants with central vision loss (simulated central scotoma studies were excluded). Studies with a comparison (before or after studies or a control group)	36 studies of which 3 were RCTs	Eccentric viewing and steady eye strategy training can improve near visual acuity, reading speed, and performance of activities of daily living. Insufficient literature to establish a relationship between training and distance visual acuity or quality of life. No conclusive evidence to show that a particular model of eccentric viewing training is superior to another and little evidence regarding the outcome and duration of training.	Most studies were judged to be of very low quality and open to risk of bias. The 3 RCTs were not well-designed studies and one confounded the effects of EV training and devices.

Authors, Year, title	Main question	Inclusion criteria	Search results	Main conclusions	Comments
Hamade et al., 2016 [179]	The effect of various low-vision rehabilitation strategies on reading speed and depression in patients 55 and older with AMD	Sample size of ≥20 eyes. RCT or observational studies from the year 2000 onwards, studies including an outcome of reading speed or depression scores	9 studies, 6 studies on reading speed (2 RCTs) and 3 on depression (2 RCTs)	Overall, a significant improvement in reading speed was found. There was a non-significant improvement in depression scores.	The number of included studies was small because reading speed was defined as the only outcome measure. The meta-analysis combined difference interventions for reading (relocation prisms, eccentric viewing training, training with devices). The risk of bias was judged as moderate to very high for all reading studies except for one, and as high for 2 of the 3 studies on depression.
Howe, 2012[124]	To compare protocols for eccentric viewing training and study factors which might predict outcomes	Broad inclusion criteria for type of study. Should include a treatment description, participants with low vision (not simulated central scotoma), outcome of reading rate.	16 studies (1 RCT). Most were before and after studies with one group.	No significant difference in reading speed based on different methods of eccentric viewing training. Eccentric viewing training is effective to improve vision. There was a negative correlation between final reading speed and age.	Some studies included other methods in addition to eccentric viewing training such as training with devices, refractive correction, and optimal lighting. This systematic review did not have a second reviewer to select the included studies.
Jutai et al., 2009[180]	For adults with low vision, what is the effectiveness of commonly prescribed assistive technology interventions for rehabilitation?	Assistive technology included optical magnification, prisms, training, telescopes, video magnifiers, illumination, computer adaptations, filters	108 studies (24 RCTs)	There were too few studies to recommend video-magnification over optical magnification. There is limited evidence comparing different prism systems for field enhancement in hemianopia. One high quality study showed that yoked prisms for relocation of the PRL in AMD are not effective. Lighting is likely to increase the benefit of optical aids for reading, but there is less evidence regarding the specific level of illumination. There is only weak evidence concerning the benefits of filters for reading. There is moderately strong evidence that people with AMD benefit from computer adaptations, specifically, size of icons.	Only 10 studies were included for detailed description.

Authors, Year, title	Main question	Inclusion criteria	Search results	Main conclusions	Comments
Liu et al., 2013[181]	The effectiveness of interventions within the scope of occupational therapy to maintain, restore and improve ADLs and IADLs at home for older adults with low vision	Not clearly stated	17 studies	Multicomponent approaches were effective. These included group sessions compared to usual care or recorded information. Single intervention was effective. Multidisciplinary low vision intervention is effective.	The single intervention studies included disparate interventions (training with devices, eccentric viewing training, prisms or full LVR). Multidisciplinary intervention studies, which compared LVR with and without an extra intervention, such as home visits, found no difference.
Rees et al., 2010 [182]	Outline the current evidence for the impact of low-vision rehabilitation programs on psychological well-being. Describe and summarize the effects of novel interventions designed specifically to address psychological needs in people with vision impairment.	Randomised, non-randomised and pre-post studies, participants 18+ years, with outcomes of mental health, anxiety, depression, self-efficacy or coping scales	30 studies (10 RCTs)	Multidisciplinary low vision rehabilitation services may improve aspects of psychological well-being such as vision specific quality of life, but has little impact on depression. Specifically designed psychological group and individual programs added to other low vision rehabilitation improved a range of psychological outcomes.	It is not clear which aspects of multidisciplinary services may improve psychological function. There are few studies which compare multidisciplinary service with optometric low vision provision.
Skelton et al., 2013[183]	The effectiveness of environmental and behavioural interventions in reducing activity limitation and improving QoL among visually impaired older people	RCTs or quasi RCTs, people 60+ years, living independently or in residential settings, studies with compared environmental interventions, behavioural interventions or both, versus control (placebo control or no intervention or usual care), or comparing different types of environmental or behavioural intervention, must have a physical activity as an outcome	No studies which met criteria	Further research is necessary to consider the effectiveness of environmental and behavioural interventions such as orientation and mobility training on physical activity, falls and quality of life in older adults with low vision, and the effect of an occupational therapist delivering home safety modification, coping strategies and exercise with older people with low vision.	

Authors, Year, title	Main question	Inclusion criteria	Search results	Main conclusions	Comments
Thomas et al., 2-15[184]	The effect of electronic assistive technologies on reading, educational outcomes and quality of life in children and young people with low vision	RCT or quasi RCTs, children and young people aged 5-16 years, studies which compare electronic devices with optical aids, studies which compared different electronic devices with each other	No studies which met criteria	High quality studies are needed to compare the usefulness of assistive technology for children and young people.	
Virgili and Rubin, 2010[185]	To assess the effects of O&M training, with or without associated devices, for adults with low vision	RCTs or quasi RCTs which compared O&M training with no training	2 small related quasi-RTCs	Low quality studies which compared training to physical exercise. Training had no significant effect.	Very strict inclusion criteria, so lower levels of evidence not assessed.
Virgili et al., 2018[108]	To assess the effects of different visual reading aids for adults with low vision	RCTs or quasi-RCTs which compared different devices for reading. Studies that compared a device with no device were excluded. Magnifying devices, filters and prisms were included.	13 studies	Reading speed may be higher with stand-mounted video magnifiers than optical devices (low certainty) and reading duration was longer with electronic devices (moderate certainty). There was less evidence for head-mounted or portable devices. No important difference between head-mounted and stand video magnifiers (low certainty) or between tablet computer and desk video magnifiers. There is no good evidence to support the use of prism relocation spectacles or coloured filters for reading. Reading speed may be decreased with coloured filters.	The authors concluded that there is insufficient evidence to support the specific type of electronic or optical device for most low vision aid users, although stand-mounted video magnifiers may improve reading speed compared to optical devices.

Appendix C

Levels of Low Vision Service

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Revised April 2019

LEVEL 1 SCREENING AND RECOGNITION OF A LV PATIENT

It is the responsibility and minimum standard of care expected of all optometrists to either directly provide Low Vision Rehabilitation (LVR), or recommend or refer for a LVR by a low vision optometrist prior to referral to other agencies. Referral should be as soon as the patient is experiencing permanent low vision, despite referrals for other treatment or on-going treatment.

Low vision assessment and rehabilitation should always be recommended for the following:

- A patient who has **low vision** which is defined as a **visual impairment** (measurable loss of vision) resulting in a **visual disability** (difficulty undertaking a task because of poor vision).

- To clarify, this includes all patients who have
  - An incurable disease or injury (ocular or systemic) for which available surgical or medical treatment has been undertaken, considered or is on-going
    AND
  - Reduced corrected vision (most commonly impairment of VA, CS or visual fields) compared to age norms
    AND
  - Difficulty with desired visual tasks despite optimum optical correction

- In terms of visual impairment, the levels at which vision loss is ***likely*** to cause a visual disability are (but not limited to) the following
  - VA 6/12 (20/40) or poorer
    OR
  - Central or paracentral scotoma or metamorphopsia
    OR
  - Peripheral field loss (hemianopia or quadrantanopia; less than 70 degrees[1] circular diameter total field)
    OR
  - Log CS < 1.4
    OR
  - A combination of these measures

Minimum additional assessment: It is important to ascertain a patient's self-reported disabilities, functional vision and goals. An accurate refraction (ideally with a trial frame) and measurement of best corrected VA are important. All optometrists should be willing and able to trial a higher reading addition (up to 4D). An assessment of contrast sensitivity and visual fields is highly recommended to complete the information required to make an accurate referral.

Minimum additional equipment: A contrast sensitivity chart such as Pelli-Robson chart, Mars Perceptrix Contrast Sensitivity Chart, Sloan Letter Low Contrast Flip Chart or the Rabin Contrast Sensitivity Test.

LEVEL 2 BASIC LV SERVICE

This level of LVR can be provided in an optometrist's office with a modest amount of equipment and optical devices, and ideally with the in-office support of a trained optometric technician/assistant or low vision therapist.

1 This includes 60 degrees which is the level for funding in Quebec

Patients who are likely to benefit are those with:

- VA from 6/12 to 6/21 inclusive and/or Log CS between 1.40 and 1.00
- No hemianopia or quadrantanopia, and circular visual field larger than 70 degrees[1]
- No significant paracentral field loss which limits reading speed or visual function

Minimum additional equipment and devices should include:

- Suitable distance acuity charts to quantify any visual acuity impairment better than HM (Bailey-Lovie chart, ETDRS chart, Feinbloom Low Vision Visual Acuity Book, Feinbloom PV numbers, Lea Numbers Low Vision Book).
- A logMAR continuous text reading acuity chart such as MNRead Chart, Colenbrander Continuous Text Near Vision Card, or Lighthouse Continuous Text
- Trial lens set for demonstration of high adds/microscopes and possibly a separate set of prism half-eyes/readers and microscopes
- Hand magnifiers (e.g., 8D, 10D, 12D, 16D) (a range of illuminated and non-illuminated, pocket-sized and larger)
- Stand magnifiers up to 4x
- Tint samples (e.g., grey, brown, yellow, orange, plum);
- Low powered monocular and binocular telescopes (e.g., up to 2-4x handheld and spectacle-mounted)
- Ideally, a good gooseneck lamp for demonstration of lighting
- Possibly a pocket video magnifier (note that patients who benefit significantly from this should be assessed for a desktop CCTV if possible, as well as other tertiary LVR)

A minimum database of necessary testing would be, but is not limited to:

a. Comprehensive history including identification of patient goals

b. Distance and near acuity testing with appropriate charts

c. Objective refraction and subjective trial frame refraction

d. Assessment of contrast sensitivity (ideally)

e. Assessment of binocularity when indicated

f. Assessment of visual fields when indicated

g. Assessment of colour vision when indicated

h. Glare assessment when indicated

i. Assessment of magnification, tint, lighting, environmental requirements

j. Development of rehabilitation plan

The optometrist should also

- Have a basic acquaintance with accessibility features on common electronic devices (iPad, cell phones, computers, tablets)
- Be able to demonstrate basic sighted guide
- Be able to discuss non-optical approaches and tips for daily living tasks and environmental modifications
- Be able to discuss issues such as driving and transportation options
- Be aware of when patients, either due to their level of vision loss, particular goals, age, or co-morbidities, require more than basic VLR.
- Be able to recognise psychological factors which may influence the adjustment to vision loss and potential for rehabilitation and refer for counselling if needed.
- Refer the patient for fully comprehensive LVR to other professionals and support organisations as indicated, for example, if the patient does not achieve his/her own goals with the LVR provided at this Level.

LEVEL 3 (COMPREHENSIVE LVR) IS ANYTHING BEYOND LEVEL 2

The Optometric LVR provider should have advanced knowledge of LVR to address complex patient presentations and provide full scope LVR. LVR at this level also includes LVR providers who are involved in multidisciplinary care, even though those LVR providers may not necessarily be in the same building.

Patients who are likely to need this level of LVR are

- VA poorer than 6/21
- CS <1.00
- Hemianopia or quadrantanopia and visual fields smaller than 70 degrees[1] circular field
- Significant central or paracentral scotoma

Minimum additional equipment and devices:

In addition to the equipment, devices and approaches listed above, the OD LVR provider would have access to a full range of

- higher levels of magnification
- complex magnification systems
  - Custom microscopes
  - Bioptics and other custom telescopes
  - Telemicroscopes
  - Head borne devices (optical and video)
  - Electro-optical magnification
- prisms
- field enhancement devices
- tints
- Lux metre for lighting measurement
- electronic magnification.

The LV optometrist should

- be able to implement eccentric viewing training, strategies for field loss,
- be familiar with support groups
- be familiar with Activities of Daily Living Skills (ADLs)
- initiate/direct patients to social assistive services (transport options, meal provision, disability tax credit registration, legal blindness registration) and make recommendations accordingly
- be capable of providing recommendations for school

The OD LVR provider should initiate appropriate referrals and communicate the rehabilitation plan, including but not limited to synopsis of exam findings, final Rx, assistive devices that are recommended and already dispensed, other device recommendations, anticipated performance with devices, training recommendations, environmental modifications, counselling and any referrals recommended or initiated.

The OD LVR provider should have working relationships with and/or refer to:

- Low vision therapist or occupational therapist
- Independent living skills provider or occupational therapist
- Orientation and mobility instructor
- High tech/CCTV/computer assessors
- Optician
- Counsellor/Psychologist
- Vision Resource/Itinerant Teachers/Teachers for the Visually Impaired
- Primary eye-care providers (referring optometrists and ophthalmologists) and other members in the patients circle of care (family physician)

Acknowledgements: Drs Julie-Andre Marinier, Alanna Stetson and Alexis Keeling for reviewing the text.

Alternative mechanisms for provision of level 3 low vision rehabilitation.[71]

<table>
<tr><th>A. Ideal – multi-disciplinary clinic (MDC) rehabilitation</th><th colspan="2">B. Second option (second most favourable) – Optometrist and vision therapist working together</th><th>C. Third option (least favourable, but maybe necessary in some locations)</th></tr>
<tr><td rowspan="12">The MDC is the ideal environment for rehabilitation of these patients[61,66] as it is generally recognised that a single profession cannot meet all the needs of people with low vision. In these clinics, vision therapists, optometrists/ ophthalmologists, opticians, O&M trainers, hi-tech assessment specialists, counsellors and others work in parallel and in the same location to create a rehabilitation plan, assess for and prescribe the full range of optical and electronic devices, address environmental modifications, train in device use, and train in sight substitution techniques.</td><td colspan="2">Since MDC are not universally available this is a second option. A vision therapist (e.g., VLRC low vision specialist) may undertake assessments in an optometrist's office or the optometrist may undertake assessments in the vision therapist / CNIB office. The assessments are undertaken in collaboration (same location, same patient visit). The way in which roles would be interrelated is shown below. Other assessments would be planned as required, e.g., O&M training, home visits.</td><td rowspan="12">The optometrist provides the initial assessment (refraction, VA, CS, fields), optical magnification, advice re lighting, filters, prisms, and training with the devices provided. The optometrist would also be involved in other training, such as eccentric viewing training and some counselling around vision loss. The optometrist refers to a vision therapist/ CNIB VLRC for other resources and rehabilitation, such as O&M, home visits, sight-substitution, training in adaptive techniques, counselling and support groups. The vision therapist/ VLRC sends a report back to the optometrist of what interventions they have undertaken. In this model, the patient may enter the system at VLRC. In this case, VLRC would request a report of visual function from the optometrist, initiate rehabilitation and then refer to the optometrist for vision devices. The optometrist would send a report back to CNIB outlining his/her interventions and recommendations.</td></tr>
<tr><td colspan="2">Optometrist and LV therapist collaborative assessment (in time sequence)</td></tr>
<tr><td>Optometrist</td><td>LV therapist</td></tr>
<tr><td></td><td>Case history/intake (goals, disabilities, current devices)</td></tr>
<tr><td>Refraction, VA (including Near VA), CS, fields</td><td rowspan="2"></td></tr>
<tr><td>Magnification estimation and suggested devices for both distance and near</td></tr>
<tr><td>Trial of spectacle-mounted devices, prisms as indicated</td><td>Trial of other magnifying optical devices, possible modification of magnification</td></tr>
<tr><td rowspan="2"></td><td>Tint trials</td></tr>
<tr><td>Assessment for video and computer devices if indicated</td></tr>
<tr><td colspan="2">Decision of recommended devices and prescription</td></tr>
<tr><td rowspan="3"></td><td>Training with recommended devices</td></tr>
<tr><td>Assessment for lighting requirements</td></tr>
<tr><td>Assessment for non-optical devices, e.g., writing aids, daily living aids, etc.</td></tr>
</table>

VLRC = Vision Loss Rehabilitation Canada, MDC: multidisciplinary clinic, O&M; orientation and mobility training

Appendix D

Estimating Magnification

MAGNIFICATION FOR NEAR

There are several ways to estimate starting magnification for near. Three common methods are described below.

1. Acuity Reserve: According to Lovie–Kitchin and Whittaker, in order for a low vision patient to read fluently, an acuity reserve needs to be considered[7]. They found that an acuity reserve of 2:1 (or a three-line difference) was effective in helping patients achieve fluent reading (approx. 100 wpm). An estimate of the required near magnification and equivalent viewing power is typically based on near word reading acuity including this acuity reserve. For adults who want to read, this is typically 2x. The target or goal print size of the patient is determined in equivalent M print (either by questioning or based on samples brought by the patient). Thus, the magnification required is 2x the ratio of the visual acuity/target print.

$$Mag = 2 * \frac{measured\ acuity\ in\ M\ print}{target\ print\ size\ in\ M\ print}$$

A typical target print size goal is 1M or the equivalent of newspaper print. For spot reading (40 wpm), a 1.3:1 (one line) minimal acuity reserve has been suggested, while 3:1 to 8.1:1 may be required for maximum reading rate.[1]

2. Critical Print Size (CPS): An alternative method to estimate required magnification is to measure the reading rate of the patient with variable print sizes. The smallest print that provides maximum reading rate is known as the critical print size.[1] In this case, there is no need to include the acuity reserve.

$$Mag = \frac{CPS\ in\ M\ print}{target\ print\ size\ in\ M\ print}$$

After the estimated magnification is calculated, the required equivalent viewing power (EVP, near addition) can then be determined:

$$EVP\ (D) = Mag\ *\ habitual\ near\ add$$

The habitual near add is the add used when the reading acuity or CPS was measured. This may also be taken as the dioptric distance used when the visual acuity or CPS was measured.

Some will calculate this requirement in terms of Equivalent Viewing Distance (the change in viewing distance that is required for the patient to meet his/her target print) and then determine the reading add or microscope required to focus at that distance.[1,186] The end result is the same.

Additionally, some patients will not have a typical habitual working distance or require magnification for a non-typical working distance. However, if the correct reading addition is in place when the habitual visual acuity and/or CPS is measured, the calculations above will still be valid.

3. Kestenbaum's Rule: This is a very quick and rudimentary way to identify the starting near addition:

$$EVP\ (D) = \frac{1}{distance\ VA}$$

Example: Distance VA = 6/18 EVP (Near Addition) = 3.00D

However, this method to calculate the near add from distance VA may underestimate the add required, as it does not include an acuity reserve and assumes that the goal is 1M print. A modification to the rule, which includes an acuity reserve would be

$$EVP\ (D) = 2 * \frac{1}{distance\ VA}$$

Estimating a reading addition for pre-presbyopic patients

1) Determine patient's age.
2) Determine patient's near acuities at habitual working distance.
3) Determine amplitude of accommodation using minimum formula (15-1/4 age)
4) Leave half of the amplitude in reserve = what patient has.
5) Determine dioptric demand at habitual working distance (what patient needs).
6) Subtract what patient has from what patient needs.
7) Example:
   10-year-old achieving 0.6M @ 10cm
   Amp = 15 – 10/4=12.5D
   Half of that is in reserve, leaving only 6.25D available for use
   Demand at 10cm is 10D
   10-6.25=3.75D= initial add to demonstrate (round to 3.5 or 4D)
8) With this add, consider if patient is reading small enough print for age and demands with an acuity reserve (at least 2x).
9) If yes, consider prescribing this add.
10) If no, calculate what additional magnification is needed to achieve the target print with an acuity reserve of 2x.
11) Aim to decrease the working distance to give this magnification and recalculate the add that you need for this new distance.
12) Example:
   Consider the 10-year-old above
   With the 3.75D add s/he obtains an acuity of 1.6M which is the required print size for this grade, i.e., there is no acuity reserve.
   A further 2x magnification is needed.
   2x magnification means bringing the print twice as close, i.e., to 5 cms (20D distance) instead of 10 cms (10D).
   Available accommodation is still 6.25D.
   Reading add for 5 cms = 20-6.25 = 13.75D = next add to demonstrate
   Alternatively, try a 2x stand magnifier with a large emergent vergence, e.g., the dome magnifier with the original add.

Clinical pearls:

- This method tends to overestimate the add – you can try reducing it.
- We would normally round up or down to the nearest diopter or half diopter.

MAGNIFICATION FOR DISTANCE

In determining the magnification for distance, it is not necessary to include an acuity reserve and typically, the target acuity can be 6/9 or 6/12 for distance and 9M or 12M at an intermediate distance.

$$Mag = \frac{denominator\ of\ patient's\ acuity}{denominator\ of\ target\ acuity}$$

when the numerators are the same.

Appendix E

Decentration and Base-in Prism Requirements for Microscopes

Both decentration and base-in prism need to be considered for patients who are binocular and prescribed microscopes for binocular viewing.

Monocular Decentration required (mms per lens)

	Add (D)				
Distance PD (mms)	+4	+6	+8	+10	+12
58	2.8	4	5.2	6.2	7.1
60	2.9	4.2	5.3	6.4	7.3
62	3.0	4.3	5.5	6.6	7.6
64	3.1	4.5	5.7	6.8	7.8
66	3.2	4.6	5.9	8.0	8.1
68	3.3	4.7	6.0	7.2	8.3

Guideline: You can also estimate this from a guideline which states that the total decentration should be 1.5mm of decentration per Dioptre of add. If the Distance PD is > 65 mm, then this rule is adjusted by adding 1mm to the total decentration, i.e., 1.5mm per Dioptre +1. This guideline gives a fairly accurate estimation. In the case of bifocals, this must be prescribed as additional inset.

Total Convergence demand in prism dioptres

	Add (D)				
Distance PD (mm)	+4	+6	+8	+10	+12
58	20.9	29.9	38.0	45.7	52.5
60	21.7	31.0	39.5	47.2	54.4
62	22.4	32.0	41.0	48.8	56.2
64	23.1	33.0	42.0	50.4	58.0
66	23.8	34.0	43.5	52.0	60.0
68	24.5	35.0	44.7	53.5	62.0

Guideline: One guideline specifies to prescribe 1 pd BI per eye for each Dioptre of add +2. For example, if prescribing a 10D add, 12pd BI per eye would be required. Note that this does NOT fully relieve the convergence demand as shown in the table above, but it is sufficient for most patients. The BI prism of prism half-eyes is according to this guideline.

Appendix F

Optics of Hand Magnifiers, Stand Magnifiers and Telemicroscopes

HAND MAGNIFIERS

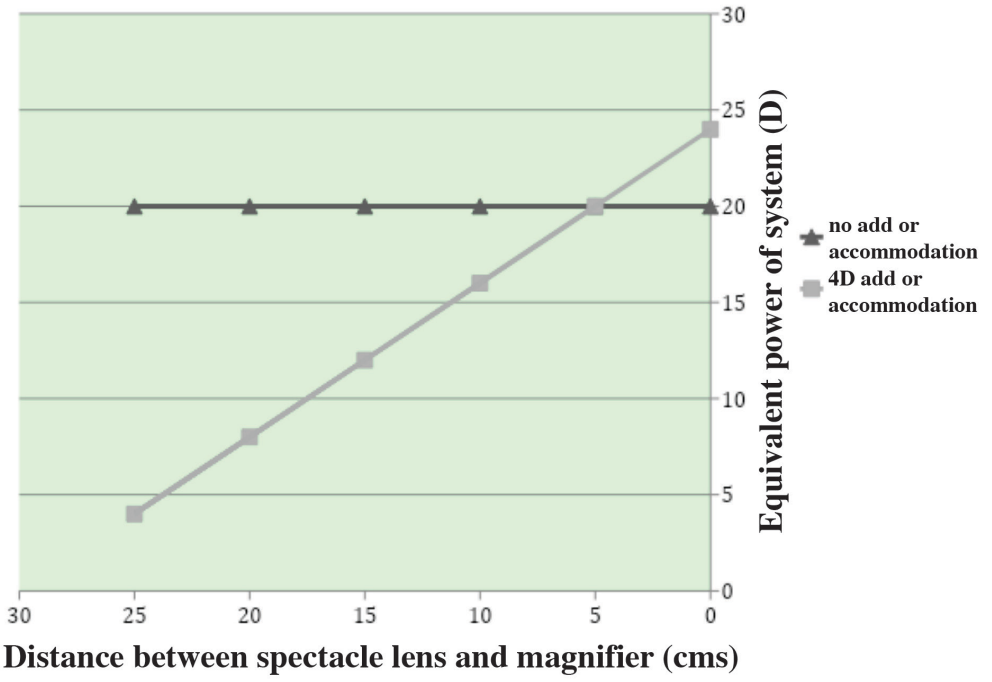
Equivalent power (EVP) obtained from a 20D hand magnifier without and with a 4D add

When a hand magnifier is used without a reading addition or accommodation, the equivalent power of the hand magnifier (and magnification) is independent of the distance between the hand magnifier and the spectacle lens (shown by triangles above).

When the hand magnifier is used with a reading addition or accommodation, an optical system of two lenses is created. The equivalent power (and magnification) of the system increases as the distance between the hand magnifier and the spectacle lens decreases (squares in the figure above). When the hand magnifier is held closer than its own focal distance, the equivalent power of the system is greater than the power of the hand magnifier itself.

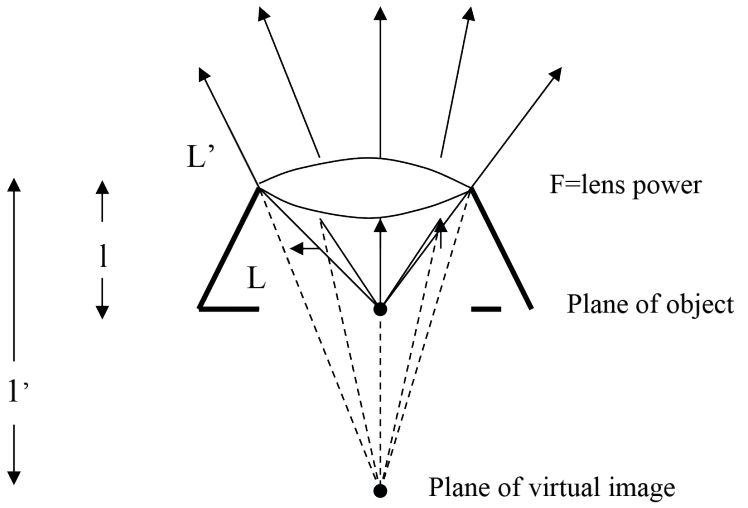
In both cases the field of view increases as the hand magnifier is brought closer to the spectacle lens.

EVP as a function of distance between the eye and the hand magnifier



STAND MAGNIFIERS

Object (L) and image (L') of a stand magnifier



where l' = image distance, l = object distance (stand height)

Transverse magnification of Stand magnifiers

The transverse magnification of a stand magnifier can be calculated from the emergent vergence (L') and the power of the lens (F). All values are entered as positive values. The manufacturer's lens powers are quite accurate and can be used in this equation. The L' must be measured or found from look-up tables.

$$T_M = \frac{L' + F}{L'}$$

Equivalent power (EVP) of stand magnifiers

$$EVP\ (D) = Reading\ add\ *\ T_M$$

where T_M is the transverse magnification of the stand magnifier

If the reading addition is to be changed, while maintaining the same EVP, the new transverse magnification is given by the relationship

$$New\ Reading\ Add * New\ T_M = Previous\ Reading\ Add * Previous\ T_M$$

so that

$$New\ T_M = \frac{Previous\ Add}{New\ Add} * Previous\ T_M$$

TELEMICROSCOPES

Equivalent viewing power (EVP) of a telemicroscope

A telemicroscope is optically composed of an afocal telescope with a positive lens (reading cap) placed on the objective lens to focus it closer than infinity. The "tele" portion is the telescope and the "microscope" portion is the reading cap. The reading cap may be a separate removable lens, or may be integrated into the objective lens.

The required EVP can be determined as described in Appendix D and the EVP of the telemicroscope (EVPTMS) is the product of the reading cap and the magnification of the afocal telescope.

$$EVP_{TMS} = F_C * M_{TS}$$

where F_C is the power of the reading cap on the telescope and MTS = the magnification of the telescope.

Alternatively, if the patient's acuity is measured at the final required viewing distance of the telemicroscope (i.e., the distance doesn't change), the magnification can be calculated as in Appendix D by the following formula:

$$Mag = 2 * \frac{measured\ acuity\ in\ M\ print}{target\ print\ size\ in\ M\ print}$$

and this will give the magnification of the telemicroscope that is required. Note that not all tasks may require a 2x acuity reserve.

Appendix G

Steps in Eccentric Viewing Training

1. Central visual field measurement to determine the size and shape of the central scotoma (see methods of visual field measurement listed in section 3.1.6 Visual Fields)

2. Determination of the probable optimal direction of eccentric fixation. This is the area of the visual field, which has the best horizontal extent to the right and which is closest to the anatomical fovea (so has best VA). This location is expected to be ideal for reading in English or other scripts which read left-to-right. The optimal direction of eccentric fixation may be different for scripts which read right-to-left or top-to-bottom.

3. Demonstration and trial with the new PRL using the Amsler charts, and samples of graded printed letters and text, such as Quillman's exercises

4. Take-home training

5. Follow-up in 2-3 weeks

6. Demonstration of steady eye strategy, whereby the eccentric viewing position is maintained and the print is scrolled through the PRL.

7. Follow-up after additional home training with assessment of magnification and prescription of devices.

Appendix H

Placement of Prisms for Peripheral Awareness in Hemianopia

SECTOR (SPOTTING PRISMS)

Fresnel prisms are placed on both lenses with their base towards the field defect (example is for right hemianopia), so that the patient does not view through the prism in the primary position, but encounters the prism when they make a small eye movement towards their hemianopia. Both prisms must be placed at the same position relative to the pupil centre, so that both are encountered with the same eye movement. For a hemianopia with no macular sparing, prisms are commonly placed halfway between the pupil edge and limbus. Typically, 20-30pd is used.

If the patient is wearing a bifocal or PAL lens, the prism is cut around the area of the near addition, so that it does not interfere with reading.

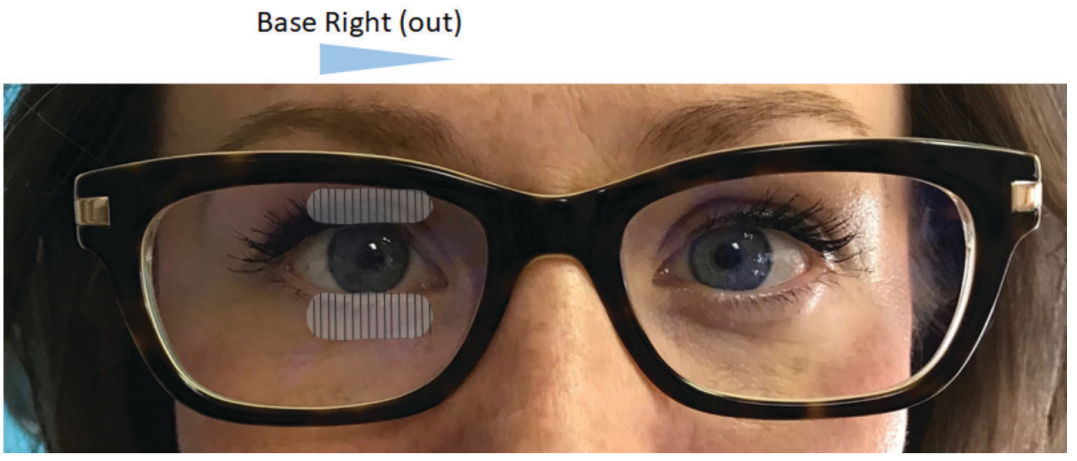
Diagrammatic representation of sector prisms for a right hemianopia

VISUAL DUPLEXING (PELI) PRISMS

These prisms are placed on the lens on the side of the hemianopia above and below fixation, with the base towards the visual field loss. The patient does not fixate through the prism, but is made aware of objects on the side of their hemianopia in their upper and lower visual field. When the patient wishes to identify an object, they turn their head to view through the central zone of the lens. The typical power is 40 pd.

As with sector prisms, the prism does not cover the bifocal area, so if the patient requires a bifocal, a small bifocal segment, such as a straight top bifocal, is placed beneath the lower prism.

Diagrammatic representation of Peli prisms for a right hemianopia



For any of these prisms, training is essential for patient success.

Appendix I

Converting Lux to Lumens

Lux required by patient	Lumens required according to Lux required by patient and anticipated distance of light source from page					
Lux required by patient (measured in LV assessment	30 cm	40 cm	50 cm	60 cm	70 cm	80 cm
500	370	660	1030	1485	2020	2640
1000	740	1320	2065	2970	4045	5285
1500	1115	1980	3100	4460	6070	7930
2000	1485	2640	4130	5945	8090	10570
2500	1860	3300	5160	7430	10116	13210
3000	2230	3965	6195	8920	12140	15855
3500	2600	4625	7225	10400	14165	18500
4000	2970	5285	8260	11890	16186	21140
4500	3345	5945	9290	13380	18210	23780
5000	3715	6605	10320	14865	20230	26425

Note: The patient's preferred Illumination in Lux is measured during the LV assessment. This table can then be used to advise the light source that the patient needs to purchase in terms of light output (Lumens). Table adapted from Borden[187]

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Conjunctival Chemosis: A Case Series of Systemic Causes

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Abstract

ABSTRACT

Conjunctival chemosis is a common ophthalmic finding that presents with a wide range of severities, symptoms, signs, and underlying etiologies. Although most cases of conjunctival chemosis are ocular in nature (allergy, infection, irritation), atypical presentations, such as dusky conjunctival hue, corkscrew conjunctival veins, and periorbital edema, should prompt further investigation for a systemic cause. In atypical cases, a review of the patient's medical history and medications, physical examination of the patient's heart and lungs, and determination of the patient's vitals (i.e., blood pressure, pulse, weight) are crucial for identifying a potential systemic source. This article reviews systemic causes of conjunctival chemosis and provides case examples to demonstrate evaluative and management techniques for optometrists to make a distinction between ocular and systemic conjunctival chemosis.

KEY WORDS:

conjunctival chemosis, periorbital edema, cutaneous, superior vena cava syndrome, hypervolemia

INTRODUCTION

Conjunctival chemosis, which is edema of the conjunctiva and the caruncle, is a common ophthalmic complication that presents with a wide range of severities, symptoms, signs, and underlying etiologies. Common clinical characteristics include diffuse translucent swelling of the bulbar conjunctiva and caruncle, folds or rugae of the conjunctival cul-de-sac, and associated tarsal conjunctival papillae.[1,2] Atypical features may include conjunctival congestion and dusky colored chemosis. To determine its etiology, it is crucial to use the ocular history, symptoms and slit lamp biomicroscopy signs (chemosis severity and color, and accompanying ocular signs). Although most cases of conjunctival chemosis are ocular in nature (allergy, infection, irritation), atypical presentations should prompt further investigation for a systemic cause. In atypical cases, a review of the patient's medical history and medications, physical examination of the patient's heart and lungs, and determination of the patient's vitals (i.e., blood pressure, pulse, weight) are crucial for identifying a potential systemic source. Systemic diseases that manifest conjunctival chemosis can be serious and even life-threatening, highlighting the importance of identifying this association. This article reviews systemic causes of conjunctival chemosis and provides case examples to demonstrate evaluative and management techniques for optometrists to distinguish between ocular and systemically derived conjunctival chemosis.

CASE REPORTS

Case 1:

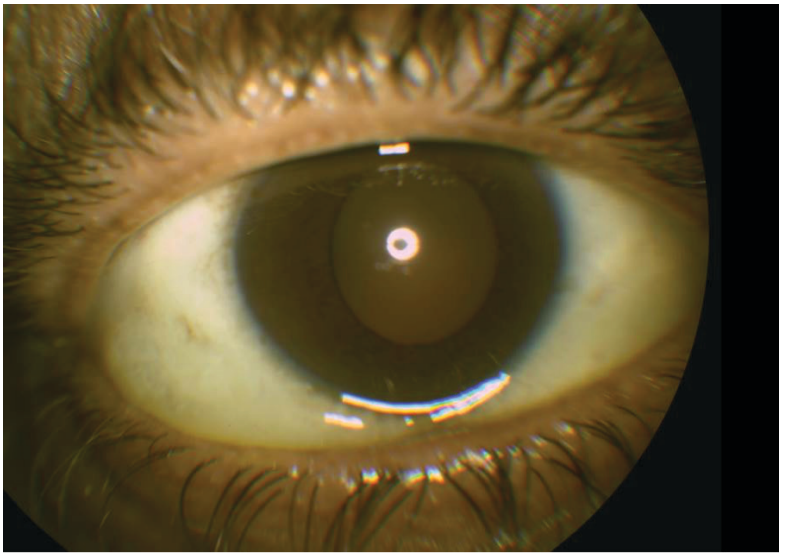
A 59-year-old African-American male reported for a routine eye exam with stable vision and good comfort OU. The patient's ocular history was pertinent for vitreous degeneration OU, blepharitis OU, and mild hypertensive retinopathy OU. He had no history of ocular trauma or surgery, recent exposure to allergens, or history of seasonal allergies. His medical history was pertinent for benign essential hypertension, atrial fibrillation without recurrence following treatment with direct current cardioversion surgery,

aneurysm of ascending aorta, bipolar affective disorder, sickle cell trait, displacement of intervertebral disc, adrenal cortical adenoma, chronic back pain, and polysubstance abuse (alcohol, marijuana, cocaine).

His medications included albuterol oral inhaler, aspirin 81mg, carvedilol, divalproex, ibuprofen, omeprazole, sildenafil citrate, and tamsulosin. Allergies included haloperidol, lurasidone, and peanuts.

Ocular examination revealed corrected visual acuities of 20/20 OD and OS. Pupils, extraocular motility, cover test and confrontation fields were normal in both eyes. Gross physical examination revealed 1+ edema of the superior and inferior orbital rim OU, venous engorgement of the right jugular vein, dyspnea, dry and non-productive cough, and pitting edema of the right and left ankles. Elevated blood pressure (160/105 mmHg) and normal pulse (84 beats per minute) were noted. Slit lamp biomicroscopy revealed eyelid collarettes. The conjunctiva revealed 3+ dusky chemosis from 6 to 9 o'clock OD and 1+ dusky pink chemosis from 3 to 4 o'clock OS (Figure 1). The cornea had arcus OU, with a tear break-up time of 10 seconds. The crystalline lens showed 1+ cortical opacities OU. Intraocular pressures by applanation were 16 mmHg OD and 14 mmHg OS at 9:55 A.M. Dilated fundus examination was significant for posterior vitreous detachment OU and tortuous retinal blood vessels consistent with hypertensive retinopathy OU.

Figure 1: *Case 1 – Diffuse conjunctival chemosis secondary to hypervolemia from acute decompensated heart failure with prolapse of bulbar conjunctiva over the lid margin*



The patient was diagnosed with atypical conjunctival chemosis. Due to his cardiac risk factors (hypertension, atrial fibrillation, tobacco use, former alcohol and illicit drug abuse), ocular signs (dusky pink conjunctival chemosis OU, periorbital edema OU), systemic signs (venous engorgement of the right jugular vein and anasarca of the right and left ankles), and symptoms (dyspnea, dry and non-productive cough), the patient was suspected to have chemosis secondary to hypervolemia due to acute decompensated heart failure. The patient was sent to the emergency department for further medical evaluation and blood work, to include renal and liver panels and cardiac biomarkers (B-type natriuretic peptide, troponin-I, and creatinine kinase). The results of the renal and liver panels were normal; however, elevated B-type natriuretic peptide levels (2,277 pg/ml) and troponin-I (0.14 ng/ml) confirmed a diagnosis of acute decompensated heart failure by cardiology, and the patient was admitted to the hospital for further evaluation and treatment. After his blood pressure was stabilized (135/90 mmHg) using intravenous hydralazine, furosemide, and acetaminophen, he was discharged from the hospital with newly prescribed atorvastatin calcium, lisinopril, and spironolactone. A cardiovascular work-up one week later, which included coronary angiogram and trans-thoracic echocardiogram, confirmed right- and left-sided heart failure, which the patient opted to medically manage with oral anti-coagulant and an anti-arrhythmic. One month later, cardiovascular stability and resolved conjunctival chemosis were confirmed by cardiology and optometry.

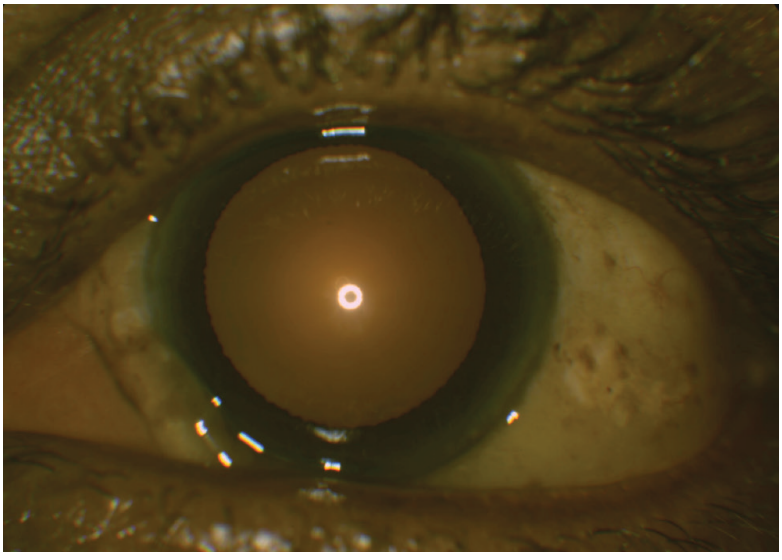
Case 2:
A 67-year-old African-American male presented for his yearly diabetic eye exam with stable vision OU. He reported itchy skin and painless left eye swelling since starting daily amlodipine 5 mg ten months previously for hypertension. His ocular history included meibomian gland dysfunction, dry eye syndrome, mild hypertensive retinopathy

OU, type 2 diabetes mellitus without ophthalmic complications OU, and mild cataracts OU. He had no history of ocular trauma or surgery, and no recent exposure to allergens. His medical history was pertinent for hypertension, hyperlipidemia, type 2 diabetes mellitus with neuropathy, obesity, sleep apnea, male erectile disorder, benign hypertrophy of the prostate, spinal stenosis of the lumbar region, osteoarthritis, and gastroesophageal reflux disease.

His medications included artificial tears, fosinopril, terazosin, simvastatin, amlodipine besylate, omeprazole, vardenafil, aspirin, tramadol, metformin, hydrochlorothiazide, and cholecalciferol (vit D3).

Ocular examination revealed corrected visual acuities of 20/20 OD and OS. Pupils, extraocular motility, cover test and confrontation fields were normal in both eyes. Gross physical examination was remarkable for peripheral pitting edema of both ankles. Mildly elevated blood pressure (162 /91 mmHg) and normal pulse (75 beats per minute) were noted. Slit lamp biomicroscopy of the eyelids revealed turbid expressions of the meibomian glands OU, conjunctival racial melanosis OU, nasal pterygium OU, and mild temporal conjunctival chalasis OD. The left eye revealed 3+ translucent bulbar conjunctival chemosis extending from the limbus to the fornix with sparing of the superior quadrant OS (Figure 2). The patient confirmed habitually sleeping in a decubitus position on his left side. The cornea, anterior chamber, iris, and lens were unremarkable OU. Tear break-up time was 7 seconds. Intraocular pressures by applanation were 15 mmHg OD and 16 mmHg OS. Dilated fundus examination revealed tortuous retinal blood vessels consistent with hypertensive retinopathy OU.

Figure 2: *Case 2 – Diffuse conjunctival chemosis secondary to amlodipine allergy with translucent conjunctival edema and caruncle swelling*



The patient was advised to continue with warm compresses b.i.d. OU and artificial tears t.i.d. OU. He was told that his conjunctival chemosis required a non-urgent consult to his primary care physician for suspected amlodipine allergy, especially after the patient confirmed itchy skin. The patient was advised to continue amlodipine, as a hypersensitivity reaction to this medication was a diagnosis of exclusion. The patient was also referred to cardiology for evaluation, due to his cardiac risk factors (diabetes, hypertension, hyperlipidemia, and sleep apnea). Cardiology evaluation with coronary angiogram, trans-thoracic echocardiogram, renal and liver panels, and cardiac biomarkers (B-type natriuretic peptide, troponin-I, and creatinine kinase) revealed ischemic heart disease without acute decompensated heart failure. Amlodipine allergy was confirmed due to cardiopulmonary stability and non-pitting anasarca of the ankles. Amlodipine was discontinued and carvedilol was initiated in its place. At the one-month follow-up cardiology and eye examination, the patient's blood pressure was well-controlled and the conjunctival chemosis was resolved.

DISCUSSION

Anatomy

During episodes of conjunctival chemosis, the bulbar conjunctiva is primarily affected. The bulbar conjunctiva is a loosely connected, semitransparent mucous membrane overlying the anterior globe between the superior and inferior fornices and extending to the corneal limbus.[3] Within the stromal layers of the bulbar conjunctiva lies a matrix of

lymphatic vessels (sub-mucosa) and blood vessels (deeper sub-mucosa) where extracellular fluids may accumulate. This space of loose attachment between the bulbar conjunctiva and underlying Tenon's layer and sclera is where fluid build-up occurs during episodes of inflammatory, infectious, or vascular events.[4] Since the palpebral conjunctiva is more tightly adherent to the tarsus, edema is less pronounced at this level and may simply present with papillae.

Ocular History

A thorough patient ocular history can be revelatory for patients who present with conjunctival chemosis since most cases have an underlying ocular etiology with a distinct mechanism of action (Table 1).[2,5-19] Common ocular etiologies include foreign body (conjunctival, corneal, eyelid), mechanical trauma (eye rubbing, blunt-force injury, trichiasis, tarsal plate concretion cysts, papillae, or follicles), acute glaucoma, ocular infection (conjunctivitis, keratitis, scleritis, endophthalmitis), or orbital infection (orbital cellulitis), allergy (conjunctivitis) and ocular inflammatory disorders (episcleritis, scleritis, uveitis).[12-15,18]

Adverse toxic effects from ophthalmic drugs, termed medicamentosa or toxic keratoconjunctivitis,[20] may also produce conjunctival chemosis. Numerous ophthalmic medications have been implicated in this type of reaction (Table 2).[16-18,21-27] Specific patterns of corneal, eyelid, and other conjunctival findings (follicular papillary reactions, symblepharon and/or fornix shortening, scarring, periocular skin hyperemia/induration/scaling) may help to confirm this etiology (Table 3).[16,17,21,28] Bulbar conjunctival injection may be greater inferiorly than superiorly, and corneal epitheliopathy may be greatest inferonasally because of increased contact time with the pharmaceutical.[28,29] Clinical signs usually take several weeks to develop[18] and unilateral or asymmetric conjunctival chemosis is not uncommon with medicamentosa or other toxic exposures.[17]

Slit Lamp Biomicroscopy Clinical Signs

Detailed slit lamp biomicroscopy is crucial for diagnosing and managing conjunctival chemosis.[1,2] The primary characteristics of conjunctival edema include translucent swelling of the bulbar conjunctiva, folds or rugae of the cul-de-sac, and associated tarsal papillae.[1] Caruncular edema presents as a swollen hyperemic caruncle, which at times can appear dry. Mild bulbar conjunctival chemosis is more likely to present as small redundant folds of conjunctiva extending past the mucocutaneous junction of the lower eyelid, while severe cases reveal prolapsed conjunctiva across the lower eyelid.[2] Although conjunctival chemosis may present unilaterally, bilateral, and symmetric involvement could indicate systemic etiology. The jelly-like conjunctival appearance is either pale and colorless, or dusky red.[1] Cases that are pale and colorless are usually due to non-venous congestion-related etiologies[1], as seen in ocular trauma, ocular infections and inflammatory conditions, chemical burns and contact hypersensitivity reactions. Blunt force and chemical ocular trauma are both ocular emergencies that require ophthalmology consultation if conjunctival chemosis is suspected to be accompanied by scleral rupture or alkaline chemical burns.[5] Conjunctival chemosis that takes on a dusky red appearance and is accompanied by prominent conjunctival venous dilation is more likely to originate from a systemic venous blockage or congestion etiology.[1] Conjunctival chemosis accompanied by corkscrew conjunctival veins suggests the possibility of retrograde venous flow, termed arterialization. Ninety percent of patients with arterialization of the conjunctival veins have carotid cavernous sinus fistulas.[30] Eyelids should be everted during slit lamp biomicroscopy to rule out a mechanical or traumatic etiology (foreign bodies, trichiasis, palpebral conjunctival and tarsal plate cysts, papillae, or follicles). It can be helpful to test corneal sensitivities if herpetic eye disease and neurotrophic keratopathy are suspected.

Symptomatology

In the absence of an underlying ocular etiology for conjunctival chemosis, a comprehensive medical history, a review of the symptomology and identification of any systemic hypersensitivity reactions to drugs, foods, chemical exposures, contact lens solution, cosmetics (skin and hair products), and illicit drugs can help determine if a systemic etiology exists (Table 4a-e[6,18,30-52] and Table 5[2,6,18,33,40,42,49,53-58]).The acuteness or chronicity of symptoms can help differentiate a localized ocular versus systemic cause of conjunctival chemosis. An acute onset of conjunctival chemosis is usually indicative of a hypersensitivity response (Table 2).[2,16-18,21-27]A subacute or chronic onset of conjunctival chemosis may have numerous etiologies , including localized ocular tissue responses (thyroid eye disease, chronic allergic conjunctivitis, ocular or eyelid surgery, trauma), increased systemic vascular permeability (allergic conditions, infections including meningitis, vasculitis), increased venous pressure (superior vena cava syndrome, heart failure), and decreased plasma oncotic pressure (nephrotic syndrome, hepatic disease).[2,18,59]

SYSTEMIC ETIOLOGIES OF CONJUNCTIVAL CHEMOSIS

Hypersensitivity Reactions

Numerous systemic medications have been implicated in hypersensitivity-related conjunctival chemosis (Table 2).[16-18,21-27] Although drug-induced hypersensitivity reactions account for 36.2% of anaphylaxis, food allergies remain the most common cause (49.7%) and recent food consumption should be investigated.[33] Ocular signs may

have concurrent dermatological signs and symptoms. Skin reactions (intense itching, flushing, eye or lip-tongue-uvula edema) and urticaria (rash sometimes accompanied by superficial dermal edema) are the most common presenting hypersensitivity symptoms, with a higher prevalence with food (90.9% and 86.9%, respectively) than with drugs (69.4% and 66.7%, respectively).[33] Although dyspnea presents equally in each group, respiratory symptoms (wheezing, stridor, hypoxemia) are more common with food allergies, while cardiovascular symptoms (syncope, hypotension, urinary incontinence, chest discomfort) are more common with drug allergies.[33]

Non-hypersensitivity Reactions

Systemic etiologies of conjunctival chemosis not attributable to hypersensitivity reactions include cavernous sinus disease (carotid cavernous sinus fistula, carotid cavernous sinus thrombosis), thyroid disease (hyperthyroidism, Grave's disease), superior vena cava syndrome, and hypervolemia (with its various underlying systemic conditions) (Table 4b-e).[6,18,30-52]

Cavernous Sinus Disease

Cavernous sinus disease (carotid cavernous sinus fistula and carotid cavernous sinus thrombosis) may present with conjunctival chemosis, in addition to numerous cranial nerve deficits.

Carotid cavernous sinus fistula is an anomalous connection between the venous system of the cavernous sinus and the internal carotid artery or its meningeal branches, or meningeal branches of the external carotid artery.[60] Conjunctival chemosis will present in 42% of patients with a carotid cavernous sinus fistula, and is characterized by the pathognomonic finding of corkscrew arterialization of the conjunctival veins (93%).[30] Additional clinical signs that occur as a result of the congestive effects of abnormal blood flow from within the cavernous sinus include proptosis (84%), decreased visual acuity (43%), cranial nerve palsy (52%), and bruit (28%).[30]

Carotid cavernous sinus thrombosis is an aseptic (surgery, trauma) or septic (sinusitis, otitis, odontogenic, facial faruncles, erysipelas) blood clot (thrombus or embolism) that travels to the cavernous sinus.[61] Acute signs presenting from congestion or blockage of the venous system include proptosis, ptosis, and chemosis.[32] These patients have a higher likelihood of a cranial nerve palsy than carotid cavernous sinus fistula patients (80-100% versus 52%), and the abducens nerve is affected most often (73%).[32] However, key differentiating symptoms of periorbital edema, headache, lethargy, and altered sensorium (50-80%) are typically observed with a cavernous sinus thrombosis.[32]

Hyperthyroidism

Hyperthyroidism can result in conjunctival chemosis secondary to fluid accumulation of glycosaminoglycans in connective tissue, resulting in edema and inflammation of the extraocular muscles, orbital connective tissue and fat, increased orbital volume, and decreased venous and lymphatic drainage.[49,53] Investigations for an associated hyperthyroid condition should account for the patient's age. Younger patients ($\leq$50 years) are classically more symptomatic than older patients ($\geq$ 70 years), but have the same core symptoms as their elders: tachycardia (96% versus 71%), fatigue (84% versus 56%), and weight loss (51% versus 50%).[33,34] Additionally, younger patients are more likely to have other symptoms, including neurological symptoms, dyspnea, and polydipsia (Table 4).[6,18,30-52] Twenty-five to forty percent of thyroid patients have thyroid orbitopathy,[35,36] with common symptoms of pain (30%), lacrimation (20.8%), diplopia at initial presentation (16.6%), photophobia (15.8%), and blurred vision (7.5%).[37]

Superior Vena Cava Syndrome

Obstruction or compression of the superior vena cava, a large vein that carries deoxygenated blood from the upper extremities, head, neck, and thorax to the right atrium, can lead to decreased venous drainage, termed superior vena cava syndrome.[40] Its most common sign is facial or neck swelling (82%)[40] that is aggravated in the horizontal position. Patients may also complain of upper-extremity swelling (68%), dyspnea (66%), and cough (50%).[40] Sixty percent of superior vena cava syndrome cases are due to malignant etiologies, specifically bronchogenic carcinoma (small cell and non-small cell lung cancer) (46%), lymphoma (8%) and germ cell tumors (8%).[40] Superior vena cava syndrome can also result from other etiologies in 40% of cases, such as medical devices (intravascular devices, dialysis catheters, pacemaker wire, Hickman catheter), and fibrosing mediastinitis (secondary to lymphoma, histoplasmosis, tuberculosis, syphilitic aortic aneurysm).[40] Seventy-one percent of these benign etiologies are secondary to intravascular medical devices, as the most common cause.[40]

Hypervolemia

Hypervolemia, also known as fluid overload syndrome, is a condition where there is excessive fluid in the blood, primarily consisting of blood plasma, salt and water. Conditions associated with hypervolemia include heart failure, nephrotic syndrome, and liver disease, each of which has its own differentiating signs and symptoms.

In the evaluation of hypervolemic heart failure patients, the physical examination has greater specificity and less sensitivity than the patient history.[41] The most common general symptom is dyspnea (87-93%), and exertional dyspnea is the most common type (86-97%).[41] Other more prevalent symptoms include edema (35-70%), jugular vein distension (5-54%), rales (25-45%) and gallop heart beat (1-26%).[41]

Nephrotic syndrome is a kidney disorder that is often idiopathic and characterized by peripheral edema, heavy proteinuria, hypoalbuminemia, and hyperlipidemia.[42-44] Type 2 diabetes mellitus and systemic lupus erythematosus are the conditions that are most commonly associated with systemic causes of nephrotic syndrome.[43,44] Clinical signs include periorbital edema that is worse in the morning, end-of-day pitting edema of the legs,[42] edema of the abdomen and genitals,[42] foamy urine secondary to proteinuria,[42] whitening of fingernails with or without white bands (Muercke's lines) secondary to severe hypoalbuminemia,[42] and skin xanthomata secondary to elevated serum cholesterol.[58] Some patients experience malaise.[42] Diabetic nephrotic syndrome patients have the classic symptoms of weight loss, polyuria, polydipsia, and polyphagia. Systemic lupus erythematosus patients most commonly present with arthritis and/or arthralgia (86%), butterfly rash (61%) and anemia (55%), followed by photosensitivity (48%), fever (43%), mouth ulcer (43%), headache (36%), the triad of fatigue, malaise, weakness (35%), and alopecia (35%).[45]

Nephrotic syndrome associated with hepatic disease may produce non-specific symptoms of malaise or abdominal discomfort[46]. Although a pertinent medical history of alcoholism can assist the optometrist with a diagnosis of fatty liver disease, non-alcoholic patients are more difficult to diagnose, as most of these cases are detected incidentally through elevated liver enzymes on routine blood examination.[46] Some patients may note an abdominal mass or a dorsocervical hump, which is associated with nonalcoholic steatohepatitis.[47]

DIFFERENTIAL DIAGNOSIS

Conjunctivochalasis and conjunctival lymphangiectasia can have features that mimic conjunctival chemosis.

Conjunctivochalasis is a bilateral aging condition in which redundant conjunctival tissue prolapses forward past the eyelid margin, possibly obstructing the lower punctae.[63] Redundant conjunctiva is usually located temporally and centrally, a few millimeters below the lower limbus,[64] while conjunctival chemosis extends to any quadrant. Mild cases of conjunctivochalasis are typically asymptomatic, while severe cases are more likely to result in punctal obstruction and tear film instability, with associated symptoms of foreign body sensation and epiphora. Such complications often result from mechanical disruption of the normal tear meniscus and impediment of nasolacrimal tear drainage.[64]

Conjunctival lymphangiectasia is a rare acquired condition that is characterized by unilateral or bilateral dilatation of the conjunctival blood vessels, usually temporarily.[65] This condition represents secondary lymphedema caused by disruption or obstruction of the conjunctival lymphatic flow.[65] It appears as diffuse enlargement of the conjunctival lymphatics (mimicking the appearance of chemosis) or focal dilatation of the lymphatics appearing as cysts. It is sometimes referred to as a "string of pearls" or "sausaging",[6] depending on the presence or absence of blood. Although the condition is most often diagnosed by appearance, recurrent cases can be biopsied.[65] Anterior segment ocular coherence tomography (OCT) can be used by clinicians to forego biopsy and confirm the presence of dilated lymphatic vessels.[66] Although the etiology is unknown, lymphangiectasia may be associated with previous trauma, other diseases (pterygium, neoplasm), or iatrogenic causes (ocular surgery, radiotherapy).[65] Lymphangiectasia patients usually complain of epibulbar irregularities including injection, irritation, and epiphora.[65]

EXTERNAL EXAMINATION

Suspicion of conjunctival chemosis as a result of a systemic condition requires physical evaluation of the patient, including the eyes and adnexa, head and neck, arms and legs, integument and hair, pulmonary system, and cardiovascular system. If a specific systemic association is suspected based upon a cursory examination, coordination of care with other specialties is important (Tables 4 and 5).[2,6,18,30-58]

Ocular Adnexa

Cases of conjunctival chemosis accompanied by eyelid retraction and edema, scleral show, and proptosis are usually indicative of hyperthyroidism. Proptosis combined with conjunctival chemosis should prompt investigations for orbital cellulitis and cavernous sinus disease, especially with deficits noted on extraocular muscle and pupillary testing.

Cardiovascular and Pulmonary Systems

Vitals (temperature, weight, pulse, blood pressure) and auscultation of the heart and lungs can provide signs (gallop, rales, wheezing, stridor) that can assist with determining a systemic etiology of chemosis.

Orbital cellulitis must be ruled out in patients who are febrile or have an oral temperature higher than 37.5°C (99.5°F). Orbital cellulitis may progress to develop signs of pain on eye movement, afferent pupillary defect, and periorbital edema.

Unintentional weight loss suggests hyperthyroidism or malignancy, while weight gain suggests hypervolemia.[41] Tachycardia could suggest hyperthyroidism or heart failure, depending on the associated signs and symptoms.[34,41] Gallop heart beat (a 3-4 beat heart sound) is heard in up to 26% of patients with heart failure, in addition to rales (crackling on inhalation) in 25-45% of patients.[41]

Hypotension (blood pressure less than 90/60 mmHg) and stridor (high-pitch whistling sound on inhalation) suggest a possible diagnosis of drug or food hypersensitivity.[33]

Dyspnea observed during external examination can be secondary to hypersensitivity reaction, hyperthyroidism, heart failure, superior vena cava syndrome, or various underlying hypervolemia etiologies.[33,40,41,44] Assessing patients for Pemberton's sign, i.e., facial congestion and cyanosis when a patient elevates both arms, is specific for latent superior vena cava syndrome.[67]

Head and Neck

Assessing the patient's head and neck is crucial to rule out lymphadenopathy and jugular vein distension, as lymphoma, thyroid, bronchogenic carcinoma, tuberculosis, systemic lupus erythematosus and syphilitic aortic aneurysm can present with the former and cardiopulmonary disease can present with the latter – all of which can be associated with systemic causes of conjunctival chemosis.

Integument and Hair

An evaluation of the patient's integument for cutaneous manifestations such as alopecia (hair loss), rashes (changes in the skin's appearance, including changes in texture and/or color), and edema can help the practitioner determine systemic etiologies of conjunctival chemosis (Table 6).[18,31,33,42,45,48,49,51,52,68-70]

Hyperthyroidism, systemic lupus erythematosus, and secondary and tertiary syphilis can cause thin and brittle hair and/or hair loss (alopecia) of the scalp, eyebrow, and eyelashes.[48,71]

Jaundice may be seen in various systemic conditions, including liver disease, tuberculosis, histoplasmosis, syphilis, Hodgkin's and non-Hodgkin's lymphoma, cardiorenal syndrome, and systemic lupus erythematosus.[51,71] Skin xanthomata are usually seen in nephrotic syndrome patients with hypercholesterolemia.[42] Patients with white nails, with or without white bands (Muercke's lines), usually have underlying nephrotic syndrome.[42] Skin rashes are more likely to be due to either a hypersensitivity reaction or systemic lupus erythematosus. Hypersensitivity reactions usually present with hives or urticaria, which appear as swollen, pale, red bumps or plaques. These reactions occur more often with foods than drugs (85.9% versus 66.7%)[33]. Systemic lupus erythematosus rashes present as a malar, "butterfly" facial rash (61%).[45]

Unilaterality, type (pitting versus non-pitting), acuteness, and location of edema on external examination can assist with determining the etiology. Patients who sleep in a lateral decubitus position may have an ipsilateral presentation of interstitial fluid accumulation, whether in a limb (arm, leg) or the conjunctiva, on the side they sleep.[52] Pitting edema, which is tissue indentation that remains after pressure is applied and released, may be observed in patients with heart failure, nephrotic syndrome, renal disease, and calcium channel blocker use.[52] Non-pitting edema, which is tissue indentation that resolves after pressure is applied and released, may be observed in lymphoma, hyperthyroidism, and superior vena cava syndrome.[52]

Acute-onset edema forming in the deeper dermal layers, termed angioedema, is usually due to hypersensitivity reactions. It can occur in any location (i.e., face, tongue, larynx, abdomen, arms, and legs), and presents more often with hypersensitivity reactions to foods (23.2%) than to drugs (18.1%).[33]

Oral edema (lip-tongue-uvula edema) accompanied by skin itching and flushing is also usually due to hypersensitivity reactions, with a higher presence in foods than drugs hypersensitivities (90.9% versus 69.4%).[33]

Periorbital edema usually presents in patients with carotid cavernous sinus thrombosis (80-100%), hypersensitivity/allergy, hyperthyroidism, and nephrotic syndrome.[32] Nephrotic syndrome patients typically present with periorbital edema that is most noticeable in the morning, end-of-day pitting edema of the legs and abdomen, and end-of-day edema of the genitals.[42]

Non-pitting pre-tibial myxedema is seen in 1-5% of Grave's disease patients.[49,52] Although pre-malar and cheek swelling is seen in hyperthyroidism patients less than 50 years of age, its presentation is rare (1.84%).[39]

Common edema noted in superior vena cava syndrome includes edema of the face or neck (82%) and upper extremities (68%), with the vast majority of these patients having bronchogenic carcinoma or the presence of an intravascular medical device.[40] The facial swelling in these lung cancer patients is prominent in the morning and resolves in the evening.

Generalized edema, anasarca, is seen in 35-70% of patients with hypervolemia.[41] It is also seen in patients with hypersensitivity reactions, like the patient in this report who developed anasarca from amlodipine.

Abdominal edema is usually secondary to nephrotic syndrome or liver disease.[43] Hepatic disease can present with abdominal, facial and peripheral edema.

Peripheral pitting edema of the arms and legs is usually due to hypervolemia (heart failure, nephrotic syndrome, liver disease).[52]

TREATMENT AND MANAGEMENT

Treatment depends on the etiology (bacterial, allergic, viral, inflammatory) and includes surgical procedures, removal of foreign bodies or eyelid cysts, and/or initiation of ophthalmic and oral antibiotics, anti-virals, anti-allergics, anti-inflammatories, and various oral and intravenous medications.

Management of conjunctival chemosis is determined by the course of the disease (i.e., acute, subacute, chronic), the etiology, and the urgency of implementing the treatment plan. Ocular etiologies usually require non-urgent coordination of care, unless the chemosis is secondary to sight-threatening ocular disorders (penetrating ocular foreign bodies, acute glaucoma, orbital cellulitis, scleral rupture, or endophthalmitis). When there is suspicion of scleral rupture or alkaline chemical burn, ophthalmology should be consulted immediately. Orbital cellulitis cases require immediate referral to the emergency department for evaluation, orbital computerized tonography with and without contrast, intravenous antibiotics, and consideration of an infectious disease referral. When all ocular etiologies are excluded, it is crucial to correlate the slit lamp biomicroscopy findings with patient history, symptoms, and external examination findings, to prevent unwarranted and expensive testing in search of systemic etiologies with a low yield (Table 3).[16,17,21,28] Chemosis that is suspected to be due to carotid cavernous sinus thrombosis, carotid cavernous sinus fistula, or acute decompensated heart failure requires immediate, same-day, referral to the emergency department for evaluation, labs, and ancillary testing (Table 4).[6,18,30-52] Non-emergent referrals to other specialties require coordination of care, so that the appropriate evaluation, labs, and ancillary testing can be completed to confirm or negate systemic conditions related to the eye-exam findings.

CONCLUSION

Conjunctival chemosis is frequently encountered in a primary eye care setting and exhibits a wide range of associations and severity. Although most cases have an ocular etiology, determined by history and examination, cases with systemic etiologies require a thorough history, review of symptomatology, physical examination (assessing for jugular vein distension, pitting and non-pitting edema, lymphadenopathy, rash, jaundice, and alopecia), and medical evaluation (pulse, BP, weight, heart and lung auscultation). In this report, two cases of conjunctival chemosis with different etiologies resulted in referrals to medical specialists to manage and treat their associated systemic conditions. ●

Table 1: *Ocular etiologies of conjunctival chemosis and corresponding mechanism of action*[2,5-19]

Etiology		Chemosis Mechanism of Action	
Ocular trauma[5,11] (including orbito-facial trauma)		Acute cases: Inflammatory reaction mediated by the release of histamine, serotonin, and bradykinin, accompanied by polymorphonuclear cell migration, which causes arteriole dilation, an increased pressure gradient between the arteriolar and venule capillaries, and extravasation of intravascular fluid and resultant vasodilatory edema[5]	Chronic cases: Blockage of lymphatic system from scarring or damage to the lymphatic drainage system[19]
Ocular surgery[7]		Blockage of lymphatic system from scarring or damage to the lymphatic drainage system[7]	
Anterior Segment Infection[6]	Conjunctivitis; Keratitis; Scleritis; Endophthalmitis (Bacterial or Viral)[6,14]	Arteriole dilation, an increased pressure gradient between the arteriolar and venule capillaries, and extravasation of intravascular fluid and resultant vasodilatory edema[18]	
Anterior Segment Inflammation[6]	Seasonal or perennial allergic conjunctivitis or keratitis[6,12]	Type I hypersensitivity (anaphylactoid) response, causing the release of histamine, serotonin, and bradykinin, accompanied by polymorphonuclear cell migration, which causes arteriole dilation, an increased pressure gradient between the arteriolar and venule capillaries, and extravasation of intravascular fluid and resultant vasodilatory edema[2,18]	
	Medicamentosa conjunctivitis or keratitis1[6,17]	Type IV hypersensitivity (cell-mediated) response where CD4 T-helper cells recognize an antigen and cause the release of interleukins, cytokines, and enzymes, resulting in the extravasation of intravascular fluid and resultant vasodilatory edema[2,18]	
	Episcleritis; Scleritis; Uveitis[8]	Arteriole dilation, an increased pressure gradient between the arteriolar and venule capillaries, and extravasation of intravascular fluid and resultant vasodilatory edema[5]	
Acute Glaucoma[23]		Numerous mechanisms depending on the etiology	
Orbital Disease[9]	Orbital infection (cellulitis)[13]	Sinus or upper respiratory infection spreading to the orbit and causing infection and inflammation of ocular tissues posterior to the orbital septum[9]	
	Orbital pseudotumor or malignancy[11]	Congestion or blockage of the lymphatic or venous system[11]	

Table 2: *Chemosis inducing ocular and systemic medications*[16-18,21-27]

Ocular Medications		Systemic Medications
Topical anesthetics	Proparacaine,[17] Tetracaine,[17] Lidocaine,[17] Benoxinate,[17] NSAIDS[17]	Birth control pills[23]
Cycloplegics	Homatropine, Atropine[17,18]	Hormone replacement[23]
Glaucoma medications	Apraclonidine,[17] Brimonidine,[17] Dorzolamide,[17] Prostaglandin analogues,[17,18] Dipivalyl epinephrine,[18] Echothiophate,[18] Pilocarpine[18]	Anti-depressants[23]
Antivirals	Idoxuridine,[18] Vidaribine,[18] Trifluoridine[18]	NSAID (ketoprofen; diclofenac)[23,25]
Aminoglycosides	Tobramycin[18] Gentamycin,[18] Neomycin,[18] Vancomycin[18]	Imatinib mesylate (Gleevac)[24]
Artificial tears	Lanolin-based artificial tears[16]	Hypertensive medications[23,26,27] (Calcium channel blockers like amlodipine)
Preservatives in ophthalmic medications	Benzalkonium chloride; phenylmercuric nitrate[17,18,22]	

Table 3: *Ocular Signs of Medicamentosa*[16,17,21,28]

Eyelid	Conjunctiva	Cornea
Punctal stenosis[21,28]	Follicular or papillary reaction[21,28]	Superficial punctate epitheliopathy[21,28]
Eyelid &/or periorbital swelling[17,21]	Bulbar injection[17,21]	Epithelial defect, ulceration[16,21]
	Symblepharon &/or fornix shortening[17,21]	Ulceration[16,28]
	Scarring[21,28]	Scarring[16]
		Pannus[16]
		Neovascularization[16]
		Perforation[16]

Table 4a: *Conjunctival chemosis with a systemic etiology of Hypersensitivity/Allergy*

Association	Presentation	Symptomology	Work-Up	Coordination of Care Specialist
Food	Acute	Skin itching, flushing, eye, lip-tongue-uvula edema (90.9%), Urticaria (86.9%), Cardiovascular symptoms such as syncope, hypotension, urinary incontinence, chest discomfort (31.3%), Hypotension (26.3%), Respiratory symptoms such as wheezing, stridor, hypoxemia (49.5%), Dyspnea (47.5%), Gastrointestinal symptoms such as crampy abdominal pain, vomiting, diarrhea (24.2%), Angioedema (23.2%), Pruritis (27.3%), and Neurological (20.2%)[33]	Skin prick test and serum allergen specific IgE tests (multiple allergosorbent test, MAST), CBC with differential, Serum total IgE, Eosinophil cationic protein (ECP), Liver panel[33]	Allergist or Emergency Department depending on the severity
Drug		Skin itching, flushing, eye, lip-tongue-uvula edema(69.4%), Urticaria(66.7%), Cardiovascular symptoms such as syncope, hypotension, urinary incontinence, chest discomfort(63.9%), Hypotension (41.7%), Respiratory symptoms such as wheezing, stridor, hypoxemia (45.8%), Dyspnea (45.8%), Gastrointestinal symptoms such as crampy abdominal pain, vomiting, diarrhea (25%), Angioedema (18.1%), Pruritis (23.6%), and Neurological (26.4%)[33]		

Table 4b: *Conjunctival chemosis with a systemic etiology of Cavernous Sinus Disease*

	Presentation	Symptomology	Work-Up	Coordination of Care Specialist
Carotid cavernous sinus fistula	Subacute or Chronic	Corkscrew arterialization of conjunctival veins (93%), Proptosis (84%), Decreased visual acuity (43%), Chemosis (42%), Ophthalmoparesis (52%: 73% abducens, 5% oculomotor, 22%multiple), and Ocular bruit (28%)[30]	CT/CTA, or MRI/MRA, or PCVD[47]	Emergency Department
Carotid cavernous sinus thrombosis	Acute	Fever, proptosis, ptosis, CN III,IV or VI palsy (80-100%), Periorbital edema, headache, lethargy, altered sensorium (50-80%), Optic disc edema, and Venous engorgement[32]	CT/CTA or MRI/MRA[58]	Emergency Department

Table 4c: *Conjunctival chemosis with a systemic etiology of Thyroid Disease (Hyperthyroidism1)*

Presentation	Symptomology	Work-Up	Coordination of Care Specialist
Subacute/ Chronic	Older patients (> 70y)[34] Tachycardia (71%), Fatigue (56%), Weight Loss (50%), Tremor (44%), Dyspnea (41%), Apathy (41%), Anorexia (32%), Nervousness (31%), Hyperactive reflexes (28%), Weakness (27%) Depression (24%), and Increased sweating (24%),[34] Alopecia,[31] Non-pitting pretibial myxedema[31,52]	Skin prick test and serum allergen specific IgE tests (multiple allergosorbent test, MAST), CBC with differential, Serum total IgE, Eosinophil cationic protein (ECP), Liver panel[33]	Allergist or Emergency Department depending on the severity
	Younger patients (< 50y)[34] Tachycardia (96%), Hyperactive reflexes (96%), Increased sweating (95%), Heat intolerance (92%), Fatigue (84%), Nervousness (84%), Tremor (84%), Polydipsia (67%), Weakness (61%), Increased appetite (57%), Dyspnea (56%), Weight Loss (51%), Diarrhea (43%), Anorexia (32%), Apathy (25%), and Depression (22%),[34] Alopecia,[31] Non-pitting pre-tibial edema (1-5%)[52]		
	Thyroid orbitopathy(25-40%)[35,36] Eyelid retraction (90.7% at some point in the disease; 70% at time of diagnosis), Exophthalmos(62%), Restrictive extraocular myopathy (43%), Optic nerve dysfunction (6%), Full constellation of manifestations including eyelid retraction, exophthalmos, optic nerve dysfunction, extraocular muscle involvement (5%), Pain (30%), Lacrimation (20.8%), Diplopia (16.6% at initial presentation) , Photophobia (15.8%), Blurred vision (7.5%), Thyroid dermopathy (4%), and Acropachy(1%),[37] Glabellar rhytids (82.5%),38 Premalar and cheek swelling(1.84%),[39] Non-pitting pre-tibial edema (1-5%),[49] Alopecia[31]		

Table 4d: *Conjunctival chemosis with a systemic etiology of Superior Vena Cava Syndrome*

	Presentation	Symptomology	Work-Up	Coordination of Care Specialist
Malignant causes (60%)[40] Lymphoma (8%)[40] Bronchogenic Carcinoma (46%)[40] • Small cell lung cancer(22%)[40] • Non-small cell lung cancer (24%)[40] Germ Cell Tumor (8%)[40] • Metastatic prostate cancer (1.2%)[40] • Thymic cancer (1.2%)[40] • Adenocarcinoma of unknown site(1.2%)[40]	Subacute/ Chronic	SVC generalized symptoms: Facial or Neck swelling (82%), Upper extremity swelling (68%), Dyspnea (66%), Cough (50%), and Dilated chest vein collaterals (38%),[40] Jaundice in lymphoma and syphillis[51]	CT or MRI chest at initial evaluation and tissue biopsy of masses[52]	Oncologist
Benign Causes(40%)[40] Intravascular device (71%), dialysis catheters (5%), pacemaker wire (1.2%), and Hickman catheter (1.2%)[40]	Subacute/ Chronic			Cardiologist
Fibrosing mediastinitis (8%)[40] • Lymphoma, Histoplasmosis, Tuberculous, and Syphilitic aortic aneurysms[40]				Infectious Disease; Cardiologist
Other (3.6%)[40] • Hematoma after aortic dissection repair; Pseudo- tumor; Primary SVC thrombosis [40]				Cardiologist

Table 4e: *Conjunctival chemosis with a systemic etiology of Hypervolemia*

	Presentation	Symptomology	Work-Up	Coordination of Care Specialist
Cardiopulmonary Failure	Subacute/ Chronic	All forms of Dyspnea, (87-93%), Exertional dyspnea (86-97%), Orthopnea(10-59%), Paroxysmal dyspnea(13-39%), Dyspnea at rest(1-6%), Weight gain (5-15%), Jugular vein distension (5-54%), Abdominal jugular reflex (6%), Edema (35-70%), Gallop heart beat (1-26%), Rales (25-45%), and Ascites (3-17%).[41] Jaundice if cardiorenal failure51	Hematocrit, protein level, albumin level, creatinine level, blood urea nitrogen level, plasma osmolality, and urine- specific gravity, Chest radiography, pulmonary artery catheterization, PRO BNP, CORS, TTE[44]	Cardiologist; Emergency Department
Nephrotic Syndrome Idiopathic (80-90%)[43,44] Primary Kidney Disease (10%)[43,44] Membranous Nephropathy (30-35%), Focal Segmental Glomerulosclerosis (30-35%), Minimal Change Disease (15%), and Immunoglobulin A nephropathy (15%)[43,44] Secondary disease (Underlying medical conditions) (10%)[43,44] Diabetes Mellitus[43,44] Immune disorders (i.e., systemic lupus erythematosus, etc.)[43,44] Infectious conditions (i.e., HIV, hepatitis, etc.)[43,44]	Subacute/ Chronic	General symptoms for primary and secondary disease: Malaise, Weight gain, Foamy urine, Morning time periorbital edema, white nails with or without white bands (Muercke's lines), skin xanthomata, and Alopecia[42] Significant evening peripheral pitting edema[31,42] Jaundice[51] Weight loss, polyuria, polydipsia, polyphagia Systemic lupus erythematosus: Arthritis and/or arthralgia (86%), butterfly rash (61%), anemia (55%), photosensitivity(48%), fever (43%), mouth ulcer (43%), headache (36%), fatigue, malaise, and weakness (35%), and alopecia(35%).[45] Non-pitting, generalized edema,.[18] Dependent pitting edema at the end of the day[18]	Urinalysis with protein to creatinine ratio, CBC and coagulation panel, renal function electrolytes, liver panel, glucose and HbA1C, focused testing for disorders by history and physical examination[43] ANA, Anti-dsDNA AB, anti-Sm AB, Anti-phospholipid AB, anti-RNA AB[42,43]	Nephrologist; Rheumatologist
Renal Disease	Subacute/ Chronic	Malaise or abdominal discomfort,[46] abdominal mass,[46] or a dorsocervical hump,[47] jaundice,[51] weight gain,[41] Facial edema, Abdominal edema, Peripheral pitting edema[52]	Renal Panel (to include AST, ALT, AST to ALT ratio, Alkaline phosphatase and γ-glutamyltransferase, bilirubin, albumin, GFR), and Hepatic Ultrasound or MRI[46]	Hepatologist

Table 5: *Conjunctival chemosis mechanism of action of systemic disorders*[2,6,18,33,40,42,49,53-58]

Etiology	Systemic Mechanism of Action		
Cavernous Sinus Disease	Carotid cavernous sinus fistula		Impaired venous drainage secondary to abnormal communication between the internal carotid artery and cavernous sinus, or the meningeal branches of the internal carotid artery or external carotid artery and cavernous sinus[57]
	Carotid cavernous sinus thrombosis		Impaired venous drainage secondary to nasal, sinus, ear, or dental infection[32,58]
Hypersensitivity/ Allergy			Allergic reaction to animal dander, pollen, food, medication, venom or drugs, mediated by the release of histamine, serotonin, and bradykinin, accompanied by polymorphonuclear cell migration, which causes arteriole dilation, an increased pressure gradient between the arteriolar and venule capillaries, and extravasation of intravascular fluid and resultant vasodilatory edema[2,18,33]
Hyperthyroidism			Fluid accumulation of glycosaminoglycans in connective tissue, resulting in edema and inflammation of the extraocular muscles, orbital connective tissue and fat, increased orbital volume, and decreased venous and lymphatic drainage[49,53]
Superior Vena Cava Syndrome	Lymphoma		Large lymph node compressing the superior vena cava causing decreased venous drainage[40]
	Bronchogenic. Carcinoma (Small and Non-Small Lung Cancer)		Mass compressing the superior vena cava causing decreased venous drainage[40]
	Germ Cell Tumor (Prostate, Thymic, Adenocarcinoma)		Mass compressing the superior vena cava causing decreased venous drainage[40]
	Intravascular devices		Device compressing the superior vena cava causing decreased venous drainage[40]
	Fibrosing mediastinitis (Lymphoma; Histoplasmosis; Tuberculosis; Syphilic aortic aneurysm)		Fibrous tissue scarring compressing the superior vena cava causing decreased venous drainage[40]
	Other (Hematoma after aortic dissection repair; Pseudo- tumor; Primary SVC thrombosis)		Compression of the superior vena cava causing decreased venous drainage[40]
Fluid Overload Syndrome	Cardiopulmonary Failure		Dilatation of the ocular vascular system caused by hypoxia, hypercapnia, which impedes thoracic venous return, by positive pressure ventilation, inducing jugular venous distention and fluid overload[56]
	Nephrotic Syndrome	Primary Kidney Disease	Low blood protein (hypoproteinemia), causing decreased colloidal osmotic pressure, which alters the osmotic gradient between the plasma and interstitial fluid, causing salt and water retention, and fluid egression into extracellular spaces[6,42,54]
		Secondary Kidney Disease, specifically Systemic Lupus Erythematosus	Patients with systemic signs: Low blood protein (hypoproteinemia), causing decreased colloidal osmotic pressure, which alters the osmotic gradient between the plasma and interstitial fluid, causing fluid egression into extracellular spaces[6,54] Patients without systemic signs: Localized edema from immune complexes, causing increased polymorphonuclear cell presence, and increased edema and inflammation[54,55]
	Liver Disease		Low blood protein (hypoalbuminemia) causing decreased colloidal osmotic pressure, an increase in oncotic pressure, and fluid egression into the ocular soft tissues[6]

Table 6: *Cutaneous presentations in systemic diseases that may present with conjunctival chemosis*[18,31,33,42,45,48,49,51,52,68-70]

Systemic Condition		Cutaneous Presentation				
		Rash	Alopecia (Yes Or No)	Jaundice (Yes Or No)	Edema (None; Pitting; Non-Pitting)	
Hypersensitivity/ Allergy	Drug-induced	Hives; Urticaria (swollen, pale, red bumps or plaques) 66.7%[33]	No	No	None	
	Food-induced	Hives; Urticaria (swollen, pale, red bumps or plaques); 86.9%[33]	No	No	None	
Hyperthyroidism/Grave's Disease		Pretibial myxedema presenting with asymptomatic warm, moist, soft, velvety, and smooth erythema to the severe elephantiasic form (1-5%)[49,52]	Yes[10]	No	Non-pitting pretibial myxedema[31,52]	
Superior Vena Cava Syndrome	Tumor, Lymph node, lymphatic vessel damage, radiotherapy[51]	No	No	Yes, if lymphoma[51]	Non-Pitting[52]	
Syphilis (presenting in extragenital areas)	Primary	Chancre without basal induration with edges rising above the surrounding surface (2%)[69]	Yes	Yes[51]	None	
	Secondary	Macular, popular, papulosquamous, pustular lesion (4.76%-59%), depending on location, with higher presentation on soles, trunk, arms, palms, legs.[70]				
	Tertiary	Firm necrotic centered gummi lesions (15%)[48]			Pitting, if patient has heart failure[52]	
Fluid Overload Syndrome	Congestive Heart Failure	No	No	Yes, if cardiorenal failure[51]	Pitting[51]	
	Nephrotic Syndrome	Primary Kidney disease	White nails with or without white bands (Muercke's lines), and skin xanthomata[42]	Yes[48]	Yes[51]	Non-pitting generalized edema[18,52] or dependent pitting edema at the end of the day[18]
		Systemic Lupus Erythematosus	White nails with or without white bands (Muercke's lines), and skin xanthomata[42] Any form of rash (>80%)[68] Butterfly rash (61%)[45]			
	Renal Disease		No	Yes[51]	Pitting[52]	

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Causes systémiques de la chimiose conjonctivale : série de cas

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RÉSUMÉ

La chimiose conjonctivale est une affection ophtalmique couramment observée qui présente divers degrés de gravité et un large éventail de symptômes, de signes et d'étiologies sous-jacentes. Bien que la plupart des cas soient de nature oculaire (allergie, infection, irritation), les manifestations atypiques comme la teinte empourprée de la conjonctive, les veines conjonctivales en tire-bouchon et l'œdème périorbital devraient inciter à chercher une cause systémique. Lorsque la chimiose est atypique, l'examen des antécédents médicaux et des médicaments du patient, l'examen physique du cœur et l'examen thoracique ainsi que la prise des signes vitaux (tension artérielle, pouls, poids) sont essentiels à l'identification de la source systémique potentielle. Cet article passe en revue les causes systémiques de la chimiose conjonctivale et présente des exemples de cas qui démontrent les techniques d'évaluation et de prise en charge permettant aux optométristes d'établir une distinction entre la chimiose conjonctivale oculaire et la chimiose conjonctivale systémique.

MOTS CLÉS :

chimiose conjonctivale, œdème périorbital, cutané, syndrome de la veine cave supérieure, hypervolémie

INTRODUCTION

La chimiose conjonctivale, caractérisée par l'œdème de la conjonctive et de la caroncule, est une complication ophtalmique courante qui présente divers degrés de gravité et un large éventail de symptômes, de signes et d'étiologies sous-jacentes. Les caractéristiques cliniques courantes comprennent l'œdème diffus et translucide de la conjonctive bulbaire et de la caroncule, l'apparition de replis ou de crêtes dans le cul-de-sac conjonctival et les papilles de la conjonctive tarsale associée.[1,2] Les caractéristiques atypiques de la chimiose peuvent comprendre la congestion ou la teinte empourprée de la conjonctive. Pour en établir l'étiologie, il est essentiel d'utiliser les antécédents oculaires, les symptômes et les signes de la biomicroscopie de la lampe à fente (gravité et coloration de la chimiose et signes oculaires connexes). Bien que la plupart des cas de chimiose conjonctivale soient de nature oculaire (allergie, infection, irritation), les manifestations atypiques devraient inciter à chercher une cause systémique. Lorsque la chimiose est atypique, l'examen des antécédents médicaux et des médicaments du patient, l'examen physique du cœur et l'examen thoracique ainsi que la prise des signes vitaux (tension artérielle, pouls, poids) sont essentiels à l'identification de la source systémique potentielle. Les maladies systémiques qui accompagnent la chimiose conjonctivale peuvent être graves et même mettre la vie en danger, ce qui souligne l'importance d'identifier cette association. Cet article passe en revue les causes systémiques de la chimiose conjonctivale et présente des exemples de cas qui démontrent les techniques d'évaluation et de prise en charge permettant aux optométristes de faire la distinction entre la chimiose conjonctivale dérivée de l'œil et la chimiose conjonctivale systémique.

RAPPORTS DE CAS

Cas 1 :

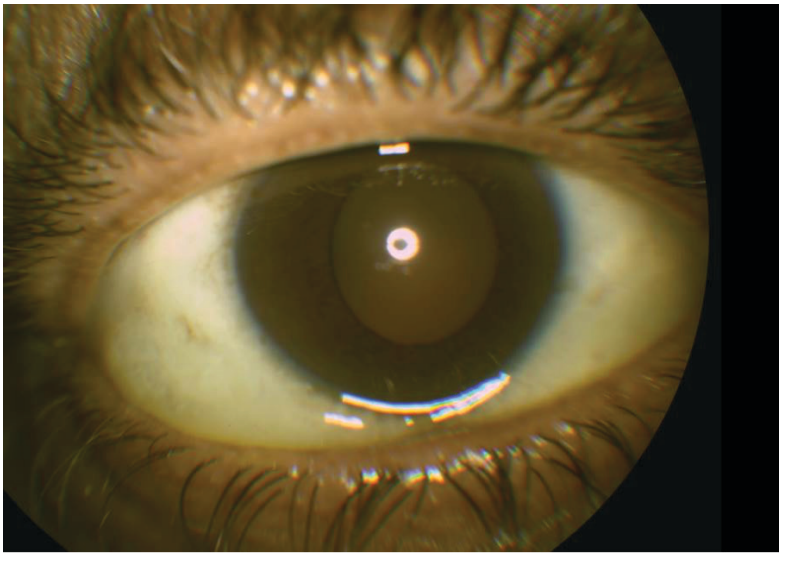
Un Afro-Américain de 59 ans s'est présenté pour un examen de la vue de routine avec une vision est stable et confortable dans les deux yeux (OU). Les antécédents oculaires du patient sont pertinents pour la dégénérescence

vitreuse OU, la blépharite OU et la rétinopathie hypertensive légère OU. Il n'y a pas d'antécédents de traumatisme oculaire ou d'intervention chirurgicale, ni d'exposition récente à des allergènes ou d'allergies saisonnières. Les antécédents médicaux sont pertinents pour l'hypertension essentielle bénigne, la fibrillation auriculaire sans récidive après le traitement par une cardioversion en courant continu, l'anévrisme de l'aorte ascendante, les troubles bipolaires, le trait drépanocytaire, le déplacement d'un disque intervertébral, l'adénome du cortex surrénal, les douleurs dorsales chroniques et la polytoxicomanie (alcool, marijuana, cocaïne).

Les médicaments comprennent l'albutérol en inhalateur oral, l'aspirine à 81 mg, le carvédilol, le divalproex, l'ibuprofène, l'oméprazole, le citrate de sildénafil et la tamsulosine. Les allergies comprennent les allergies à l'halopéridol, à la lurasidone et aux arachides.

L'examen oculaire a révélé une acuité visuelle corrigée de 20/20 OD et OS. Les pupilles, la motilité extraoculaire, le test de l'écran et la périmétrie par confrontation étaient normaux dans les deux yeux. L'examen physique complet a révélé un œdème de 1+ du bord orbital supérieur et inférieur, l'engorgement veineux de la veine jugulaire droite, la dyspnée, la toux sèche sans expectoration et l'œdème retenant le godet de la cheville droite et de la cheville gauche. On a noté une tension artérielle élevée (160/105 mm Hg) et un pouls normal (84 battements par minute). La biomicroscopie de la lampe à fente a révélé des collerettes sur les paupières. La conjonctive a révélé une chimiose avec teinte empourprée de 3+ de 6 à 9 heures OD et une chimiose rose foncé de 1+ de 3 à 4 heures OS (figure 1). Un arc cornéen OU a été observé avec un temps de bris du film lacrymal de 10 secondes. Le cristallin présentait une opacité corticale de 1+ OU. Les pressions d'aplanation intraoculaires étaient de 16 mm Hg OD et de 14 mm Hg OS à 9 h 55. L'examen du fond d'œil dilaté a révélé un décollement postérieur de la rétine OU et des vaisseaux sanguins rétiniens tortueux correspondant à une rétinopathie hypertensive OU.

Figure 1 : *Cas 1 : Chimiose conjonctivale diffuse secondaire à l'hypervolémie causée par une insuffisance cardiaque aiguë en décompensation avec prolapsus de conjonctive bulbaire sur la marge de la paupière*



Le patient a reçu un diagnostic de chimiose conjonctivale atypique. En raison des facteurs de risque de cardiopathie chez ce patient (hypertension, fibrillation auriculaire, tabagisme, consommation antérieure d'alcool et de drogues illicites), de signes oculaires (chimiose avec coloration rosée de la conjonctive OU, œdème périorbital OU), des signes d'affection systémique (engorgement veineux de la jugulaire droite et anasarque des chevilles gauche et droite) et des symptômes (dyspnée, toux sèche sans expectoration), une chimiose secondaire à une hypervolémie causée par une insuffisance cardiaque en décompensation a été soupçonnée. Le patient a été envoyé à l'urgence pour qu'il subisse un examen médical plus poussé et des analyses sanguines, y compris une série de tests de la fonction rénale et hépatique et des biomarqueurs cardiaques (peptide natriurétique de type B, troponine-I et créatinine kinase). Les résultats de la série de tests de la fonction rénale et hépatique étaient normaux. Toutefois, des niveaux élevés de peptide natriurétique de type B (2 277 pg/ml) et de troponine-I (0,14 pg/ml) ont confirmé un diagnostic d'insuffisance cardiaque décompensée aiguë par un cardiologue. Le patient a été admis à l'hôpital pour une évaluation et un traitement plus poussés. Après que l'on ait stabilisé la tension artérielle du patient (135/90 mm Hg) à

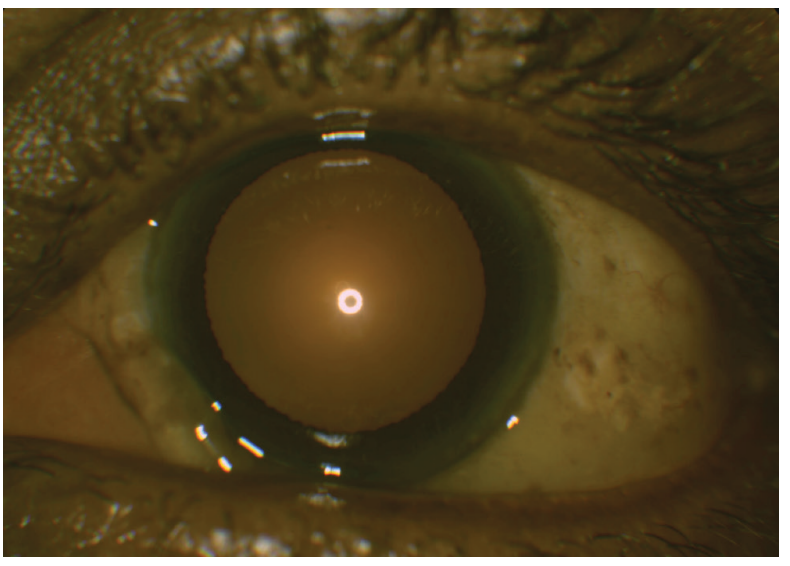
l'aide d'hydralazine intraveineuse, de furosémide et d'acétaminophène, celui-ci a reçu son congé de l'hôpital et une nouvelle prescription d'atorvastatine calcique, de lisinopril et de spironolactone. Un bilan cardiovasculaire effectué une semaine plus tard, lequel comprenait un angiogramme coronarien et un échocardiogramme transthoracique, a confirmé une insuffisance cardiaque droite et gauche que le patient a choisi de gérer médicalement avec un anticoagulant oral et un antiarythmique. Un mois plus tard, des tests de cardiologie et d'optométrie ont confirmé la stabilité cardiovasculaire et la disparition de la chimiose conjonctivale.

Cas 2 :
Un Afro-Américain diabétique de 67 ans s'est présenté à son examen annuel de la vue avec une vision stable OU. Il a dit éprouver des démangeaisons de la peau et une enflure indolore de l'œil gauche depuis dix mois, soit depuis qu'il prend 5 mg d'amlodipine tous les jours pour traiter l'hypertension. Ses antécédents oculaires comprennent un dysfonctionnement meibomien, le syndrome de l'œil sec, une légère rétinopathie hypertensive OU, le diabète sucré de type 2 sans complications ophtalmiques OU et de légères cataractes OU. Il n'y a pas d'antécédents de traumatisme oculaire ou d'intervention chirurgicale ni d'exposition récente à des allergènes. Les antécédents médicaux sont pertinents pour l'hypertension, l'hyperlipidémie, le diabète sucré de type 2 avec neuropathie, l'obésité, l'apnée du sommeil, le trouble érectile masculin, l'hypertrophie bénigne de la prostate, la sténose rachidienne de la région lombaire, l'arthrose et le reflux gastro-œsophagien.

Les médicaments du patient comprennent des larmes artificielles, le fosinopril, la térazosine, ls simvastatine, le bésylate d'amlodipine, l'oméprazole, le vardénafil, l'aspirine, le tramadol, la métformine, l'hydrochlorothiazide et cholécalciférol (vitamine D3).

L'examen oculaire a révélé une acuité visuelle corrigée de 20/20 OD et OS. Les pupilles, la motilité extraoculaire, le test de l'écran et la périmétrie par confrontation étaient normaux dans les deux yeux. L'examen physique général a permis de relever un œdème périphérique retenant le godet dans les deux chevilles. Une tension artérielle légèrement élevée (162/91 mm Hg) et un pouls normal (75 battements par minute) ont été notés. La biomicroscopie de la lampe à fente des paupières a révélé des sécrétions troubles des glandes de Meibomius OU, une mélanose conjonctivale d'origine raciale OU, un ptérygion nasal OU et une légère chalasise conjonctivale OD. L'œil gauche a révélé une chimiose conjonctivale bulbaire translucide de 3+ allant du limbe au fornix avec conservation du quadrant supérieur OS (figure 2). Le patient a confirmé qu'il dormait habituellement en décubitus sur son côté gauche. La cornée, la chambre antérieure, l'iris et le cristallin OU n'avaient rien d'anormal. Le temps de bris du film lacrymal était de 7 secondes. Les pressions d'aplanation intraoculaires étaient de 15 mm Hg OD et de 16 mm Hg OS. L'examen du fond de l'œil dilaté a révélé la présence de vaisseaux sanguins rétiniens tortueux correspondant à une rétinopathie hypertensive OU.

Figure 2 : *Cas 2 : Chimiose conjonctivale diffuse secondaire à une allergie à l'amlodipine avec œdème conjonctival translucide et enflure de la caroncule*



On a conseillé au patient de continuer à appliquer des compresses chaudes 2 f.p.j. OU et des larmes artificielles 3 f.p.j. OU. On lui a dit que sa chimiose conjonctivale nécessitait une consultation non urgente chez un médecin de premier recours en raison d'une allergie présumée à l'amlodipine, surtout après que le patient ait confirmé ressentir des démangeaisons cutanées. On a conseillé au patient de continuer à prendre de l'amlodipine car une réaction d'hypersensibilité à ce médicament aurait constitué un diagnostic d'exclusion. Le patient a également été aiguillé vers un cardiologue pour subir une évaluation en raison de ses facteurs de risque cardiaques (diabète, hypertension, hyperlipidémie et apnée du sommeil). L'évaluation du cardiologue, qui comprenait un angiogramme coronarien, un échocardiogramme transthoracique, une série de tests de la fonction rénale et hépatique et des biomarqueurs cardiaques (peptide natriurétique de type B, troponine-I et créatinine kinase) a révélé une cardiopathie ischémique sans insuffisance cardiaque décompensée aiguë. L'allergie à l'amlodipine a été confirmée en raison de la stabilité cardiopulmonaire et de l'anasarque des chevilles ne retenant pas le godet. L'amlodipine a été abandonnée et remplacée par le carvédilol. Lors du suivi à un mois en cardiologie et de l'examen de la vue, la tension artérielle du patient était bien contrôlée et la chimiose conjonctivale avait disparu.

DISCUSSION

Anatomie

Pendant les épisodes de chimiose conjonctivale, la conjonctive bulbaire est principalement touchée. La conjonctive bulbaire est une muqueuse semitransparente et lâchement rattachée qui recouvre le globe antérieur, entre le fornix supérieur et inférieur, et qui rejoint le limbe cornéen[3]. Dans les couches du stroma de la conjonctive bulbaire se trouve une matrice de vaisseaux lymphatiques (sous-muqueuse) et de vaisseaux sanguins (sous-muqueuse profonde) où peuvent s'accumuler les liquides extracellulaires. L'espace entre la conjonctive et le fascia de Ténon, où l'attachementt est lâche, est le lieu où les fluides s'accumulent durant l'inflammation, l'infection et les épisodes vasculaires[4]. Comme la conjonctive palpébrale adhère fermement au tarse, l'œdème est moins prononcé à ce niveau et peut simplement affecter les papilles.

Antécédents oculaires

Un examen approfondi des antécédents oculaires peut être révélateur chez les patients qui présentent une chimiose conjonctivale du fait que la plupart des cas ont une étiologie oculaire sous-jacente ayant un mécanisme d'action distinct (tableau 1)[2,5-19]. Les étiologies oculaires courantes comprennent les corps étrangers (conjonctive, cornée, paupières), les traumatismes mécaniques (frottement des yeux, choc violent, trichiasis, kyste dans la concrétion du tarse, papilles ou follicules), glaucome aigu, infection oculaire (conjonctivite, kératite, sclérite, endophthalmite) ou infection orbitale (cellulite orbitaire), allergie (conjonctivite) et autres troubles oculaires inflammatoires (épisclérite, sclérite, uvéite)[12-15,18].

Les effets toxiques indésirables des médicaments ophtalmiques, appelés « medicamentosa » ou « kérato-conjonctivite »[20], peuvent aussi produire une chimiose conjonctivale. De nombreux médicaments ophtalmique engendrent ce type de réaction (tableau 2)[16-18,21-27]. Des schémas particuliers d'atteintes de la cornée, des paupières ou de la conjonctive (réactions folliculaires papillaires, amenuisement du symblépharon et/ou du fornix, scarification, hyperémie/induration/écaillage de la peau périoculaire) peuvent contribuer à confirmer cette étiologie (tableau 3)[16,17,21,28]. Une injection dans la conjonctive bulbaire peut s'avérer plus efficace dans la région inférieure que supérieure et une épithéliopathie de la cornée peut mieux réagir au niveau inféronasal en raison de la durée du contact avec les substances pharmaceutiques[28,29]. Les signes cliniques prennent habituellement plusieurs semaines à se développer[18] et la chimiose conjonctivale unilatérale ou asymétrique n'est pas rare en présence d'une medicamentosa ou autre exposition toxique[17].

Biomicroscopie de la lampe à fente – signes cliniques

La biomicroscopie détaillée des lampes à fente est essentielle au diagnostic et à la prise en charge la chimiose conjonctivale.[1,2] Les principales caractéristiques de l'œdème conjonctival comprennent l'œdème translucide de la conjonctive bulbaire, l'apparition de replis ou de crêtes dans le cul-de-sac conjonctival et les papilles de la conjonctive tarsale associé[1]. L'œdème de la caroncule se manifeste par une hyperémie de la caroncule, laquelle paraît parfois sèche. La chimiose conjonctivale bulbaire légère est plus susceptible de présenter des replis et des crêtes répétitifs qui s'étendent au-delà de la jonction mucocutanée de la paupière inférieure, tandis que les cas graves révèlent une conjonctive prolabée sur la paupière inférieure[2]. Bien que la chimiose conjonctivale puisse se manifester unilatéralement, une atteinte bilatérale et symétrique pourrait indiquer une étiologie systémique. La conjonctive a un aspect gélatineux, elle est soit pâle soit incolore, soit rougeâtre[1]. Les chimioses conjonctivales de couleur pâle ou incolores sont habituellement attribuables à des étiologies liées à une congestion non veineuse[1], comme on le voit dans les traumatismes oculaires,

les infections oculaires et les conditions inflammatoires, les brûlures chimiques et les réactions d'hypersensibilité de contact. Les chocs violents et les traumatismes chimiques sont des urgences oculaires qui nécessitent une consultation en ophtalmologie lorsqu'une chimiose conjonctivale est soupçonnée d'être accompagnée d'une rupture de la conjonctive scléroticale ou d'une brûlure chimique alcaline[5]. La chimiose conjonctivale qui prend une teinte rougeâtre et qui s'accompagne d'une dilatation veineuse importante est plus susceptible d'être causée par un blocage veineux systémique ou une congestion[1]. La chimiose conjonctivale montrant des veines en tire-bouchon laisse supposer un écoulement veineux rétrograde appelé « artérialisation ». Quatre-vingt-dix pour cent des patients qui présentent une artérialisation des veines conjonctivales ont une fistule des sinus caverneux carotidiens[30]. Les paupières doivent être retournées pendant la biomicroscopie avec lampe à fente pour exclure une étiologie mécanique ou traumatique (corps étrangers, trichiasis, kyste dans la concrétion du tarse et de la conjonctive palpébrale, papilles ou follicules). Il peut être utile de vérifier les sensibilités cornéennes si l'on soupçonne un herpès oculaire et une kératopathie neurotrophique.

Symptomatologie

En l'absence d'une étiologie oculaire sous-jacente à la chimiose conjonctivale, les antécédents complets du patient, l'examen de la symptomatologie et l'identification de toute réaction d'hypersensibilité aux médicaments, aux aliments, aux produits chimiques, aux solutions pour lentilles cornéennes, aux produits cosmétiques (pour la peau et les cheveux) ainsi que de la consommation de drogues illicites peuvent aider à établir si l'étiologie est de nature systémique (tableau 4a-e [6,18,30-52] et tableau 5[2,6,18,33,40,42,49,53-58].)

L'acuité ou la chronicité des symptômes peut aider à différencier la cause localisée de la chimiose conjonctivale oculaire de la cause systémique. L'apparition soudaine d'une chimiose conjonctivale est habituellement révélatrice d'une réaction d'hypersensibilité (tableau 2)[2,16-18,21-27]. L'apparition d'une chimiose conjonctivale sous-aiguë ou chronique peut avoir de nombreuses étiologies, incluant une réaction localisée des tissus oculaires (thyroïdopathie affectant les yeux, conjonctivite consécutive à une allergie chronique, intervention chirurgicale visant les paupières ou les yeux, traumatisme), hausse de la perméabilité vasculaire systémique (états allergiques, infections comprenant la méningite, vascularite), hausse de la pression veineuse (syndrome de la veine cave supérieure, insuffisance cardiaque) et baisse de la pression oncotique plasmatique (syndrome néphrotique, néphropathie)[2,18,59].

ÉTIOLOGIES SYSTÉMIQUES DE LA CHIMIOSE CONJONCTIVALE

Réactions d'hypersensibilité

De nombreux médicaments systémiques ont été impliqués dans la chimiose conjonctivale liée à l'hypersensibilité (tableau 2)[16-18,21-27]. Bien que les réactions d'hypersensibilité d'origine médicamenteuse représentent 36,2 % de l'anaphylaxie, les allergies alimentaires demeurent la cause la plus courante (49,7 %) et la consommation récente d'aliments doit être examinée[33]. Les signes oculaires peuvent présenter des signes et des symptômes dermatologiques concomitants. Les réactions cutanées (démangeaisons intenses , bouffées vasomotrices, œdème des yeux, des lèvres, de la langue et de la luette) et l'urticaire (éruptions cutanées parfois accompagnées d'œdème dermique superficiel) montrent les symptômes d'hypersensibilité les plus courants. Leur prévalence est plus élevée quand elles sont associées aux aliments (90,9 % et 86,9 %, respectivement) par rapport aux médicaments (69,4 % et 66,7 %, respectivement)[33]. Bien que les manifestations de dyspnée soient égales dans les deux groupes, les symptômes respiratoires (respiration sifflante, stridor, hypoxémie) sont plus courants en présence d'une allergie alimentaire tandis que les symptômes cardiovasculaires (syncope, hypotension, incontinence urinaire, malaise pulmonaire) sont plus courants en présence d'une allergie aux médicaments[33].

Réactions non attribuables à une hypersensibilité

Les étiologies systémiques de la chimiose conjonctivale qui ne sont pas attribuables aux réactions d'hypersensibilité comprennent les atteintes du sinus caverneux (fistule ou thrombose carotido-caverneuse), la thyroïdopathie (hyperthyroïdie, maladie de Graves), le syndrome de la veine cave supérieure et l'hypervolémie [avec ses diverses conditions systémiques sous-jacentes] (tableau 4b-e)[6,18,30-52].

Atteintes du sinus caverneux

Une atteinte du sinus caverneux (fistule ou thrombose carotido-caverneuse) peut se présenter avec une chimiose conjonctivale, en plus de nombreux déficits des nerfs crâniens.

La fistule carotido-caverneuse est une communication anormale entre le système veineux du sinus caverneux et l'artère carotide interne ou ses ramifications vers les méninges ou, encore, dans les ramifications de l'artère carotide externe vers les méninges[60]. La chimiose conjonctivale est manifeste chez 42 % des patients ayant une fistule carotido-caverneuse et se caractérise par l'observation pathognomonique d'une artérialisation en tire-bouchon des

veines conjonctivales (93 %)[30]. D'autres signes cliniques attribuables à l'effet de congestion de la circulation sanguine anormale dans le sinus caverneux comprennent l'exophtalmie (84 %), la baisse de l'acuité visuelle (43 %), la paralysie des nerfs crâniaux (52 %) et le souffle (28 %)[30].

La thrombose carotido-caverneuse est un caillot de sang (thrombus ou embolie) d'origine aseptique (intervention chirurgicale, traumatisme) ou septique (sinusite, otite, infection œdontogène, furoncle facial, érysipèle) qui se déplace vers les sinus caverneux[61]. Les signes d'atteinte aiguë émanant d'une congestion ou du blocage du système veineux comprennent l'exophtalmie, la ptosis et la chimiose[32]. Ces patients sont plus susceptibles d'avoir une paralysie des nerfs crâniens que les patients ayant une fistule carotido-caverneuse patients (80-100 % contre 52 %) et le nerf abducens est affecté la plupart du temps (73 %)[32]. Cependant, les principaux symptômes de différenciations que sont l'œdème périorbital, les maux de tête, la léthargie et les troubles de la conscience (50-80 %) sont des effets types de la thrombose carotido-caverneuse[32].

Hyperthyroïdie

L'hyperthyroïdie peut entraîner une chimiose conjonctivale secondaire à l'accumulation de glycosaminoglycanes dans les tissus conjonctifs, ce qui entraîne un œdème et une inflammation des muscles extraoculaires, des tissus conjonctifs orbitaux et des tissus adipeux, une augmentation du volume orbital ainsi qu'une diminution du drainage veineux et lymphatique[49,53]. La recherche d'une hyperthyroïdie associée devrait tenir compte de l'âge du patient. Les patients plus jeunes (≤ 50 ans) ressentent habituellement davantage de symptômes que les patients plus âgés (≥ 70 ans), bien que les symptômes types soient les mêmes dans les deux groupes : tachycardie (96 % contre 71 %), fatigue (84 % contre 56 %) et perte pondérale (51 % contre 50%)[33,34]. En outre, les patients plus jeunes sont plus susceptibles d'éprouver d'autres symptômes dont des symptômes neurologiques, la dyspnée et la polydipsie (tableau 4)[6,18,30-52]. Vingt-cinq pour cent des patients atteints de thyroïdopathie ont une orbitopathie d'origine thyroïdienne[35,36] et éprouvent les symptômes courants suivants : douleur (30 %), larmoiement (20,8 %), diplopie lors de la manifestation initiale (16,6 %), photophobie (15,8 %) et vision brouillée (7,5 %)[37].

Syndrome de la veine cave supérieure

L'obstruction ou la compression de la veine cave supérieure, grande veine dans laquelle le sang désoxygéné transite des extrémités des membres supérieurs, de la tête, de la nuque et du thorax jusqu'au ventricule droit, peut affaiblir le drainage veineux, état appelé « syndrome de la veine cave supérieure »[40]. L'un de ses signes les plus courants est l'œdème du visage ou de la nuque (82 %)[40], lequel s'aggrave en décubitus. Les patients mentionnent parfois aussi une enflure des extrémités des membres supérieurs (68 %), de la dyspnée (66 %) et de la toux (50 %)[40]. Soixante pour cent des cas de syndrome de la veine cave supérieure ont une étiologie de malignité, plus précisément un cancer broncho-pulmonaire (à petites cellules ou non à petites cellules) (46 %), un lymphome (8 %) ou des tumeurs de cellules germinales (8%)[40]. Le syndrome de la veine cave supérieure a d'autres étiologies dans 40 % des cas, par exemple le matériel médical (dispositifs intravasculaires, cathéters de dialyse, fils de stimulateurs cardiaques, cathéter Hickman) et la médiastinite fibreuse (secondaire à un lymphome, à l'histoplasmose, à la tuberculose, à un anévrisme aortique d'origine syphilitique)[40]. Soixante et onze pour cent de ces étiologies bénignes sont secondaires à l'utilisation d'un dispositif médical intravasculaire, laquelle constitue la cause la plus courante[40].

Hypervolémie

L'hypervolémie, qui se traduit par une surcharge liquidienne, est une affection où le sang contient un excès de liquides, principalement du plasma sanguin, du sel et de l'eau. Les affections associées à l'hypervolémie comprennent l'insuffisance cardiaque, le syndrome néphrotique et les maladies hépatiques, chacune ayant des signes et des symptômes distinctifs.

Lors de l'évaluation des patients atteints d'insuffisance cardiaque hypervolémique, l'examen physique a une spécificité supérieure et sensibilité moindre que les antécédents du patient[41]. Le symptôme général le plus courant est la (87-93 %) dont la dyspnée d'effort est le type le plus courant (86-97 %)[41]. D'autres symptômes plus prévalents comprennent l'œdème (35-70 %), la distension de la veine jugulaire (5-54 %), les râles (25-45 %) et le bruit de galop du cœur (1-26 %)[41].

Le syndrome néphrotique est un trouble rénal souvent idiopathique et caractérisé par de l'œdème périphérique, une forte protéinurie, de l'hypoalbuminémie et de l'hyperlipidémie[42-44]. Le diabète sucré de type 2 et le lupus érythémateux systémique sont des affections les plus souvent associées aux causes systémiques du syndrome néphrotique[43,44]. Les signes cliniques comprennent l'œdème périorbital, plus accentué le matin, l'œdème des jambes retenant le godet en fin de journée[42], l'œdème de l'abdomen et des parties génitales[42], l'urine spumeuse attribuable à une protéinurie[42], la coloration blanche des ongles avec ou sans bandes blanches (lignes de Muehrcke) attribuable à une hypoalbuminémie[42] et les xanthomes cutanés attribuables à un taux élevé de cholestérol sérique[58]. Certains patients éprouvent un malaise[42]. Les patients diabétiques atteints de syndrome néphrotique présentent les symptômes clas-

siques que sont la perte pondérale, la polyurie, la polydipsie et la polyphagie. Les patients atteints de lupus érythémateux systémique sont le plus souvent atteints d'arthrite et/ou d'arthralgie (86 %), d'érythème en papillon (61 %) et d'anémie (55 %), suivis de la photosensibilité (48 %), de la fièvre (43 %), d'ulcères de la bouche (43 %), de maux de tête (36 %), de la triade de la fatigue, des malaises et de la faiblesse (35 %) et d'alopécie (35 %)[45].

Le syndrome néphrotique associé à une néphropathie peut causer des symptômes non spécifiques de malaise ou d'inconfort abdominal[46]. Bien que l'examen pertinent des antécédents d'alcoolisme puisse aider l'optométriste à établir un diagnostic de stéatose hépatique, les patients non alcooliques sont plus difficiles à diagnostiquer du fait que la plupart de ces cas sont détectés de manière fortuite lorsque le taux d'enzymes hépatiques est élevé ou au moyen des tests sanguins habituels[46]. Certains patients peuvent avoir noté la présence d'une masse abdominale ou d'un renflement dorso-cervical, lesquels sont associés à une stéatose hépatique non alcoolique[47].

DIAGNOSTIC DIFFÉRENTIEL

La chalasise conjonctivale et la lymphangiectasie conjonctivale peuvent avoir des caractéristiques qui ressemblent à celles d'une chimiose conjonctivale.

La chalasise conjonctivale est une atteinte bilatérale associée au vieillissement dans laquelle des prolapsus de tissu conjonctival redondant se forment vers l'avant, au-delà de la marge de la paupière, possiblement en obstruant le méat lacrymal inférieur[63]. La conjonctive redondante est habituellement localisée vers les tempes et le centre, quelques millimètres sous le limbe inférieur[64], tandis que la chimiose conjonctivale s'étend à n'importe quel quadrant. Les cas bénins de chalasise conjonctivale sont généralement asymptomatiques, tandis que les cas graves sont plus susceptibles d'entraîner une obstruction du méat lacrymal et une instabilité du film lacrymal associées à la sensation d'avoir un corps étranger et d'un épiphore. Ces complications résultent souvent d'une perturbation mécanique du ménisque lacrymal et de l'altération du drainage nasolacrymal[64].

La chalasise conjonctivale est une maladie acquise rare caractérisée par une dilatation unilatérale ou bilatérale des vaisseaux sanguins de la conjonctive habituellement temporaire[65]. Cet état représente un lymphœdème secondaire causé par la rupture ou l'obstruction du flux lymphatique dans la conjonctive[65]. Elle se manifeste par l'élargissement diffus des vaisseaux lymphatiques de la conjonctive (qui donnent l'apparence d'une chimiose) ou par la dilatation focalisée des vaisseaux qui ressemblent alors à des kystes. On parle parfois de « chaîne de perles » ou de « saucissons »[6], selon la présence ou l'absence de sang. Bien que le diagnostic de cet état soit le plus souvent basé sur l'apparence, il convient de pratiquer une biopsie quand il devient récurrent[65]. Une tomographie par cohérence optique du segment antérieur peut aider les cliniciens à éviter la biopsie et confirmer la présence de vaisseaux lymphatiques dilatés[66]. Bien que l'étiologie soit inconnue, la lymphangiectasie peut être associée à un traumatisme antérieur, à d'autres maladies (ptérygion, néoplasme) ou à des causes iatrogènes (intervention chirurgicale oculaire, radiothérapie)[65]. Les patients atteints de lymphangiectasie se plaignent habituellement d'irrégularités épibulbaires comprenant l'injection, l'irritation et l'épiphore[65].

EXAMEN EXTERNE

Une chimiose conjonctivale soupçonnée attribuable à un problème systémique nécessite l'examen physique du patient, y compris des yeux et de leurs annexes, de la tête et de la nuque, des bras et des jambes, du système tégumentaire et des cheveux, du système pulmonaire et de l'appareil cardiovasculaire. Si l'on soupçonne une association systémique particulière à la suite d'un examen superficiel, la coordination des soins avec d'autres spécialistes est importante (tableaux 4 et 5)[2,6,18,30-58].

Annexes de l'œil

Les cas de chimiose conjonctivale accompagnée d'une rétraction de la paupière et d'œdème, de tissu scléral apparent et d'une exophtalmie sont habituellement révélateurs d'une hyperthyroïdie. La combinaison d'une exophtalmie et d'une la chimiose conjonctivale devrait mener à la recherche d'une cellulite orbitale et du syndrome des sinus caverneux, en particulier si des déficits sont observés lors des tests du muscle extraoculaire et de la pupille.

Appareil cardiovasculaire et système pulmonaire

Les signes vitaux (température, poids, pouls, tension artérielle) et l'auscultation du cœur et des poumons sont des signes (bruit de galop, râles, respiration sifflante, stridor) qui peuvent aider à établir l'étiologie systémique de la chimiose.

La cellulite orbitale doit être exclue chez les patients fébriles ou dont la température orale est supérieure à 37,5 °C (99,5 °F). La cellulite orbitale peut progresser et causer des signes de douleur lors du mouvement oculaire, un déficit pupillaire afférent et un œdème périorbital.

Lae perte de poids involontaire est indicatrice d'une hyperthyroïdie ou d'une malignité tandis que la prise de poids suggère une hypervolémie[41].

La tachycardie pourrait indiquer une hyperthyroïdie ou une insuffisance cardiaque, selon les signes et symptômes connexes[34,41]. Le bruit de galop (rythme cardiaque à 3 ou 4 temps) est perçu chez jusqu'à 26 % des patients qui ont une insuffisance cardiaque, en plus des râles (crépitement à l'inhalation) chez 25 à 45 % des patients[41].

L'hypotension (tension artérielle inférieure à 90/60 mm Hg) et le stridor (bruit de sifflement aigu lors de l'inhalation) évoquent un diagnostic possible d'hypersensibilité aux médicaments ou aux aliments[33].

La dyspnée observée lors de l'examen externe peut être secondaire à une réaction d'hypersensibilité, à l'hyperthyroïdie, à l'insuffisance cardiaque, au syndrome de la veine cave supérieure ou à diverses étiologies d'hypervolémie sous-jacentes[33,40,41,44]. L'évaluation du signe de Pemberton chez les patients, c.-à-d. l'apparition d'une congestion faciale et d'une cyanose lorsqu'un patient élève les deux bras, est spécifique d'un syndrome latent du syndrome de la veine cave supérieure[67].

Tête et nuque

Il est essentiel d'évaluer la tête et la nuque du patient pour écarter la possibilité d'une lymphadénopathie et d'une distension des veines jugulaires du fait que les lymphomes, les thyroïdopathies, les cancers bronchopulmonaires, la tuberculose, le lupus érythémateux systémique et les anévrismes aortiques d'origine syphilitique peuvent être associés à une lymphadénopathie et une maladie cardiopulmonaire à la distension des veines jugulaires. Tous ces symptômes peuvent se manifester en présence d'une chimiose conjonctivale d'étiologie systémique.

Système tégumentaire et cheveux

L'examen du système tégumentaire du patient pour déceler des manifestations cutanées comme l'alopécie (perte de cheveux), les éruptions cutanées (changements de l'apparence de la peau, y compris de la texture et/ou de la couleur) et l'œdème peut aider le praticien à déterminer les étiologies systémiques de la chimiose conjonctivale (tableau 6)[18,31,33,42,45,48,49,51,52,68-70].

L'hyperthyroïdie, le lupus érythémateux systémique et la syphilis secondaire et tertiaire peuvent rendre les cheveux épars et cassants et/ou causer une perte de cheveux (alopécie) du cuir chevelu, des sourcils et des cils[48,71].

La jaunisse peut être présente avec diverses pathologies systémiques, notamment les maladies hépatiques, la tuberculose, l'histoplasmose, la syphilis, les lymphomes hodgkiniens et non hodgkiniens, le syndrome cardio-rénal et le lupus érythémateux systémique[51,71]. Les xanthomes cutanés sont habituellement observés chez les patients ayant un syndrome néphrotique et de l'hypercholestérolémie[42]. Les patients dont les ongles sont de couleur blanchâtre, avec ou sans bandes blanches (lignes de Muehrcke), ont habituellement un syndrome néphrotique sous-jacent[42]. Les éruptions cutanées sont plus susceptibles d'être attribuables à une réaction d'hypersensibilité ou à un lupus érythémateux systémique. Les réactions d'hypersensibilité se manifestent habituellement par de l'urticaire qui forme des bosses rougeâtres ou des plaques pâles accompagnées d'œdème. Ces réactions sont plus souvent associées aux aliments qu'aux médicaments (85,9 % contre 66,7 %)[33]. Les éruptions en forme de papillon du lupus érythémateux systémique sont présentes dans les régions malaires du visage (61 %)[45].

L'unilatéralité, le type (retenant le godet ou non), l'acuité et l'emplacement de l'œdème déterminés lors de l'examen externe peuvent aider à établir l'étiologie. Les patients qui dorment en position allongée latérale peuvent présenter, sur le côté où ils se couchent, une accumulation ipsilatérale de liquides interstitiels, par exemple dans un bras ou dans une jambe ou, encore, dans la conjonctive[52]. Un œdème retenant le godet, défini par l'indentation des tissus qui se prolonge après l'application et le retrait d'une pression, peut être observé chez les patients souffrant d'insuffisance cardiaque, de syndrome néphrotique et de néphropathie ou qui prennent des inhibiteurs calciques[52]. L'œdème ne retenant pas le godet, défini par l'indentation des tissus qui se résorbe après l'application et le retrait d'une pression, peut être observé chez les patients souffrant d'un lymphome, d'hyperthyroïdie et du syndrome de la veine cave supérieure[52].

L'apparition soudaine d'un œdème dans les couches dermiques plus profondes, appelé œdème angioneurotique, est habituellement attribuable à une réaction d'hypersensibilité. Il peut apparaître n'importe où (c.-à-d. sur le visage, la langue, le larynx, l'abdomen, les bras et les jambes) et est plus souvent associé à une réaction d'hypersensibilité aux aliments (23,2 %) qu'à une réaction aux médicaments (18,1 %)[33].

L'œdème buccal (de la langue, des lèvres et de la luette) accompagné de démangeaisons et de bouffées vasomotrices est aussi généralement dû à une réaction d'hypersensibilité; il est plus souvent présent dans les réactions d'hypersensibilité associées aux aliments que dans celles associées aux médicaments (90,9 % contre 69,4 %)[33].

Un œdème périorbital est habituellement observable chez les patients qui ont une thrombose carotido-caverneuse (80 à 100 %), une hypersensibilité ou une allergie, une hyperthyroïdie ou un syndrome néphrotique[32]. Les patients atteints du syndrome néphrotique présentent habituellement un œdème périorbital plus notable le matin, un œdème des jambes et de l'abdomen retenant le godet en fin de journée un œdème des parties génitales en fin de journée[42].

Chez 1 à 5 % des patients atteints de la maladie de Graves, on observe un myxœdème prétibial ne retenant pas le godet[49,52]. Bien que l'on observe une enflure prémalaire et de la joue chez les patients de moins de 50 ans atteints d'hyperthyroïdie, cette manifestation est rare (1,84 %)[39].

L'œdème commun observé avec le syndrome de la veine cave supérieure comprend l'œdème du visage et de la nuque (82%) et des extrémités des membres supérieurs (68%), la grande majorité des patients atteints ayant un cancer bronchopulmonaire ou un dispositif médical intravasculaire[40]. L'œdème du visage chez les patients atteints de cancer du poumon est prononcé le matin et se résorbe en soirée.

Un œdème généralisé, l'anasarque, est observé chez 35 à 70 % des patients atteints d'hypervolémie[41]. On l'observe également chez les patients qui ont une réaction d'hypersensibilité, comme c'est le cas du patient chez qui l'amlodipine a provoqué une anasarque.

L'œdème abdominal est habituellement secondaire au syndrome néphrotique ou à une néphropathie[43]. Une néphropathie peut se présenter avec un œdème abdominal, facial et périphérique.

L'œdème périphérique des bras et des jambes est habituellement attribuable à une hypervolémie (insuffisance cardiaque, syndrome néphrotique, affection hépatique)[52].

TRAITEMENT ET PRISE EN CHARGE

Le traitement dépend de l'étiologie (bactérienne, allergique, virale, inflammatoire) et comprend des interventions chirurgicales, le retrait de corps étrangers ou de kystes dans la paupière et/ou l'amorçage d'un traitement par des antibiotiques ophtalmiques et oraux, des antiviraux, des antiallergiques, des anti-inflammatoires et divers médicaments oraux et intraveineux.

La prise en charge de la chimiose conjonctivale est déterminée par l'évolution de la maladie (c.-à-d. aiguë, subaiguë, chronique), l'étiologie et l'urgence de mettre en œuvre le plan de traitement. Les étiologies oculaires nécessitent habituellement une coordination des soins non urgente, à moins que la chimiose soit secondaire aux troubles oculaires menaçant la vue (pénétration de corps étrangers oculaires, glaucome aigu, cellulite orbitale, rupture de la sclérotique ou énophtalmie). Lorsqu'on soupçonne une rupture de la sclérotique ou une brûlure chimique alcaline, il faut consulter sans tarder un ophtalmologiste. Les cas de cellulite orbitale requièrent le renvoi immédiat du patient vers le service des urgences pour évaluer son état et lui faire subir une tomographie orbitale avec ou sans contraste et où on lui administrera des antibiotiques intraveineux. Il pourrait aussi être redirigé vers un infectiologue. Quand toutes les étiologies oculaires sont exclues, il s'avère crucial de corréler les résultats de la biomicroscopie de la lampe à fente avec les antécédents du patient, ses symptômes et les résultats de l'examen externe. Cela évite la réalisation de tests coûteux dont les résultats ne sont pas garantis ni nécessairement révélateurs pour trouver une étiologie systémique (tableau 3)[16,17,21,28]. Une chimiose que l'on pense attribuable à une thrombose ou à une fistule carotido-caverneuse ou, encore, à une insuffisance cardiaque aiguë en décompensation nécessite le renvoi immédiat et le jour même du patient au service des urgences pour lui faire subir une évaluation, des tests de laboratoire et des tests connexes (tableau 4)[6,18,30-52]. Les renvois non très urgents vers d'autres spécialistes nécessitent une coordination des soins pour qu'une évaluation, des tests de laboratoire et des tests connexes appropriés soient effectués afin de confirmer ou d'infirmer les états systémiques liés aux résultats de l'examen de la vue.

CONCLUSION

La chimiose conjonctivale est fréquente dans un contexte de soins oculovisuels primaires et présente un large éventail d'associations et divers degrés de gravité. Bien que la plupart des cas aient une étiologie oculaire, déterminée par les antécédents et l'examen, ceux dont l'étiologie est systémique exigent l'examen approfondi des antécédents et de la symptomatologie, un examen physique (évaluation de la distension de la veine jugulaire, de l'œdème retenant ou ne retenant pas le godet, de la lymphadénopathie, des éruptions cutanées, de la jaunisse et de l'alopécie) et une évaluation médicale (pouls, TA, poids, auscultation du cœur et du thorax). Dans ce rapport, deux cas de chimiose conjonctivale avec des étiologies différentes ont mené à un aiguillage vers des médecins spécialistes pour gérer et traiter les problèmes systémiques associés. ●

Tableau 1 : *Étiologies oculaires de la chimiose conjonctivale et mécanismes d'action correspondants* [2,5-19]

Étiologie		Mécanisme d'action de la Chimiose	
Traumatisme oculaire[5,11] (y compris un traumatisme orbito-facial)		*Aiguë :* Réaction inflammatoire provoquée par la libération de l'histamine, de la sérotonine et de la bradykinine accompagnée d'une migration des cellules polymorphonucléaires, laquelle provoque la dilatation des artérioles, la hausse du gradient de pression entre les capillaires artériolaires et les veinules ainsi que l'extravasation du liquide intravasculaire et œdème de vasodilatation consécutif[5]	*Chronique :* Blocage du système lymphatique attribuable à une scarification ou à une altération du drainage lymphatique[19]
Chirurgie oculaire[7]		Blocage du système lymphatique attribuable à une scarification ou à une altération du drainage lymphatique[7]	
Infection du segment antérieur[6]	Conjonctivite; kératite; sclérite; énophtalmie (bactérienne ou virale)[6,14]	Dilatation des artérioles, hausse du gradient de pression entre les capillaires artériolaires et les veinules et extravasation du liquide intravasculaire et œdème vasodilataire consécutif[18]	
Inflammation du segment antérieur[6]	Conjonctivite allergique saisonnière ou permanente ou kératite[6,12]	Réaction d'hypersensibilité de type I (anaphylactoïde) entraînant une libération d'histamine, de sérotonine et de bradykinine, accompagnée d'une migration de cellules polymorphonucléaires, laquelle provoque la dilatation des artérioles, la hausse du gradient de pression entre les capillaires artériolaires et les veinules ainsi que l'extravasation du liquide intravasculaire et un œdème de vasodilatation consécutif[2,18]	
	Conjonctivite medicamentosa ou kératite[16,17]	Réponse d'hypersensibilité de type IV (à médiation cellulaire) où les lymphocytes T auxiliaires (CD4) reconnaissent un antigène et déclenchent la libération d'interleukines, de cytokines et d'enzymes, ce qui entraîne l'extravasation de fluides intravasculaires et un œdème de vasodilatation consécutif[2,18]	
	Épisclérite; sclérite; uvéite[8]	Dilatation des artérioles, hausse du gradient de pression entre les capillaires artériolaires et les veinules et extravasation du liquide intravasculaire et œdème vasodilataire consécutif[5]	
Glaucome aigu[23]		Nombreux mécanismes selon l'étiologie	
Maladie orbitale[9]	Infection orbitale (cellulite)[13]	Infection des sinus ou des voies respiratoires supérieures se propageant vers l'orbite et causant une infection et une inflammation des tissus oculaires postérieurs à la cloison orbitale[9]	
	Pseudotumeur ou malignité orbitale[11]	Congestion ou blocage du système lymphatique ou veineux[11]	

Tableau 2 : *Médicaments oculaires et systémiques qui induisent des chimioses*[16-18,21-27]

Médicaments Oculaires		Médicaments Systémiques
Anesthésiques topiques	Proparacaïne[17] Tétracaïne[17] Lidocaïne[17] Benoxinate[17] AINS[17]	Pilules contraceptives[23]
Cycloplégiques	Homatropine, atropine[17,18]	Hormonothérapie[23]
Médicaments contre le glaucome	Apraclonidine[17] Brimonidine[17] Dorzolamide[17] Analogues des prostaglandine[17,18] Dipivalyle épinéphrine [18] Échothiophate[18] Pilocarpine[18]	Antidépesseurs[23]
Antiviraux	Idoxuridine[18] Vidaribine[18] Trifluoridine[18]	AINS (kétoprofène; diclofénac)[23,25]
Aminosides	Tobramycine[18] Gentamycine[18] Néomycine[18] Vancomycine[18]	Mésylate d'imatinib (Gleevec)[24]
Larmes artificielles	Larmes artificielles à base de lanoline[16]	Anti-hypertenseurs[23,26,27] (inhibiteurs calciques comme l'amlodipine)
Agents de conservation des médicaments ophtalmiques	Chlorure de benzalkonium; nitrate phénylmercurique [17,18,22]	

Tableau 3 : *Signes oculaires de medicamentosa* [16,17,21,28]

Paupière	Conjonctive	Cornée
Sténose ponctuelle[21,28]	Réaction folliculaire ou papillaire[21,28]	Épithéliopathie ponctuée superficielle[21,28]
Œdème oculaire et/ou périorbital[17,21]	Injection bulbaire[17,21]	Trouble épithélial, ulcération [16,21]
	Amenuisement du symblépharon et/ou du fornix[17,21]	Ulcération16,[28]
	Scarification[21,28]	Scarification[16]
		Pannus[16]
		Néovascularisation[16]
		Perforation[16]

Tableau 4a : *Chimiose conjonctivale avec étiologie systémique d'hypersensibilité/d'allergie*

Association	Présentation	Symptomatologie	Bilan	Coordination du Spécialiste de la Santé
Aliment	Aiguë	Démangeaisons cutanées, bouffées vasomotrices, œdème de l'œil, des lèvres, de la langue et de la luette (90,9 %), urticaire (86,9 %), symptômes cardiovasculaires comme la syncope, l'hypotension, l'incontinence urinaire, les douleurs thoraciques (31,3 %), l'hypotension (26,3 %), les symptômes respiratoires comme la respiration sifflante, le stridor, l'hypoxémie (49,5 %), la dyspnée (47,5 %), les symptômes gastro-intestinaux comme les douleurs et crampes abdominales, les vomissements, la diarrhée (24,2 %), l'œdème angioneurotique (23,2 %), le prurit (27,3 %) et les symptômes neurologiques (20,2 %)[33].	Test de la piqûre cutanée et tests de détection sérique des IgE spécifiques d'allergènes (test à allergosorbant multiple, MAST), hémogramme avec analyse différentielle, IgE sériques totales, protéine cationique de l'éosinophile, série de tests de la fonction hépatique[33]	Allergologue ou service des urgences selon la gravité
Médicament		Démangeaisons cutanées, bouffées vasomotrices, œdème de l'œil, des lèvres, de la langue et de la luette (69,4 %), urticaire (66,7 %), symptômes cardiovasculaires comme la syncope, l'hypotension, l'incontinence urinaire, les douleurs thoraciques (63,9 %), l'hypotension (41,7 %), les symptômes respiratoires comme la respiration sifflante, le stridor, l'hypoxémie (45,8 %), la dyspnée (45,8 %), les symptômes gastro-intestinaux comme les douleurs et crampes abdominales, les vomissements, la diarrhée (25 %), l'œdème angioneurotique (18,1 %), le prurit (23 %) et les symptômes neurologiques (26,4 %)[33].		

Tableau 4b : *Chimiose conjonctivale avec étiologie systémique du syndrome des sinus caverneux*

	Présentation	Symptomatologie	Bilan	Coordination du Spécialiste de la Santé
Fistule carotido-caverneuse	Subaiguë ou chronique	L'artérialisation en tire-bouchon des veines conjonctivales (93 %), l'exophtalmie (84 %), la diminution de l'acuité visuelle (43 %), la chimiose (42 %), ophthalmoparésie (52 % : 73 % de l'abducteur, 5 % oculomoteur, 22 % multiple) et bruit oculaire (28 %)[30]	Tomodensitométrie / angiographie par tomodensitométrie, ou imagerie par résonance magnétique / angiographie par résonance magnétique, ou densité des vaisseaux capillaires péripapillaires (PCVD)[47]	Service des urgences
Thrombose carotido-caverneuse	Aiguë	Fièvre, exophtalmie, ptose, paralysie des nerfs crâniens III, IV ou VI (80-100 %), œdème périorbital, maux de tête, léthargie, troubles de la conscience (50-80 %), œdème périorbital et engorgement veineux[32]	Tomodensitométrie / angiographie par tomodensitométrie ou imagerie par résonance magnétique / angiographie par résonance magnétique[58]	Service des urgences

Tableau 4c : *Chimiose conjonctivale avec étiologie systémique de trouble thyroïdien (hyperthyroïdie 1)*

Présentation	Symptomatologie	Bilan	Coordination du Spécialiste de la Santé
Sous-aiguë/ chronique	*Patients plus âgés (> 70 ans)*[34] Tachycardie (71 %), fatigue (56 %), perte pondérale (50 %), tremblements (44 %), dyspnée (41 %), apathie (41 %), anorexie (32 %), nervosité (31 %), réflexes hyperactifs (28 %), faiblesse (27 %), dépression (24 %) et sudation accrue (24 %)[34], alopécie[31] et myxœdème ne retenant pas le godet[31,52]	Test de la piqûre cutanée et tests de détection sérique des IgE spécifiques d'allergènes (test à allergosorbant multiple, MAST), hémogramme avec analyse différentielle, IgE sériques totales, protéine cationique de l'éosinophile, série de tests de la fonction hépatique[33]	Allergologue ou service des urgences selon la gravité
	Patients plus jeunes (< 50 ans)[34] Tachycardie (96 %), réflexes hyperactifs (96 %), sudation accrue (95 %), intolérance à la chaleur (92 %), fatigue (84 %), nervosité (84 %), tremblement (84 %), polydipsie (67 %), faiblesse (61 %), augmentation de l'appétit (57 %), dyspnée (56 %), perte pondérale (51 %), diarrhée (43 %), anorexie (32 %), apathie (25 %) et dépression (22 %)[34], alopécie[31], œdème prétibial ne retenant pas le godet (1-5 %)[52]		
	Orbitopathie d'origine thyroïdienne (25-40 %)[35,36] Rétraction de la paupière (90,7 % à un moment ou à un autre de la maladie; 70 % lors du diagnostic), exophtalmie (62 %), myopathie extraoculaire restrictive (43 %), dysfonction du nerf optique (6 %), constellation de manifestations comprenant la rétraction de la paupière, l'exophtalmie, la dysfonction du nerf optique, un trouble du muscle extraoculaire (5 %), la douleur (30 %), le larmoiement (20,8 %), une diplopie 16,6 lors de la manifestation initiale) , la photophobie (15,8 %), la vision trouble (7,5 %), une dermopathie d'origine thyroïdienne (4 %) et une acropathie (1 %)[37], ridules de la glabelle (82,5 %)[38], enflure de la région prémalaire et des joues (1,84 %)[39], œdème prétibial ne retenant pas le godet (1-5 %)[49], alopécie[31]		

Tableau 4d : *Chimiose conjonctivale avec étiologie systémique du syndrome de la veine cave supérieure*

	Présentation	Symptomatologie	Bilan	Coordination du Spécialiste de la Santé
Causes malignes (60 %)40 Lymphome (8 %)[40] Carcinome bronchogène (46 %)[40] • Cancer du poumon à petites cellules (22 %)[40] • Cancer du poumon non à petites cellules (24 %)[40] Tumeur des cellules germinales (8 %)[40] • Cancer métastatique de la prostate (1,2 %)[40] • Cancer du thymus (1,2 %)[40] • Adénocarcinome d'un site inconnu (1,2 %)[40]	Sous-aiguë/ chronique	Symptômes généralisés du SVC : Enflure du visage ou de la nuque (82 %), enflure des extrémités des membres supérieurs (68 %), dyspnée (66 %), toux (50 %) et dilatation des veines collatérales thoraciques (38 %)[40], jaunisse dans le lymphome et syphilis[51]	TDM ou IRM thoracique à l'évaluation initiale et biopsie tissulaire des masses[52]	Oncologue
Causes bénignes (40 %)40 Appareil intravasculaire (71 %), cathéters de dialyse (5 %), fil de stimulateur cardiaque (1,2 %) et cathéter de Hickman (1,2 %)[40]	Sous-aiguë/ chronique			Cardiologue
Médastinite fibreuse (8 %)[40] • Lymphome, histoplasmose, tuberculose et anévrisme de l'aorte syphilitique[40]				Maladie infectieuse; cardiologue
Autres (3,6 %)[40] • Traitement de l'hématome après une dissection aortique; pseudotumeur; thrombose primaire de la VCS[40]				Cardiologue

Tableau 4e : *Chimiose conjonctivale avec étiologie systémique d'hypervolémie*

	Présentation	Symptomatologie	Bilan	Coordination du Spécialiste de la Santé
Défaillance cardiorespiratoire	Sous-aiguë/ chronique	Toutes les formes de dyspnée (87-93 %), de dyspnée d'effort (86-97 %), d'orthopnée (10-59 %), de dyspnée paroxysmale (13-39 %), de dyspnée au repos (1-6 %), de gain pondéral(5-15 %), de distension de la veine jugulaire (5-54 %), de réflexe jugulaire abdominal (6 %), d'œdème (35-70 %), de bruit de galop du cœur (1-26 %), de râles (25-45 %) et d'ascite (3-17 %)[41]. Jaunisse en cas de défaillance cardio-rénale[51]	Hématocrite, taux de protéines, taux d'albumine, taux de créatinine, taux d'azote uréique dans le sang, osmolité plasmatique et densité de l'urine, radiographie thoracique, cathétérisation de l'artère pulmonaire, PRO BNP, tests cardiaques, échocardiographie transthoracique[44]	Cardiologue; service des urgences
Syndrome néphrotique Idiopathique (80-90 %)[43,44] Néphropathie primaire (10 %)[43,44] Néphropathie membraneuse (30-35 %), glomérulosclérose segmentale et focale (30-35 %), syndrome néphrotique à lésions glomérulaires minimes (15 %) et néphropathie à immunoglobuline A (15 %)[43,44] Maladie secondaire (affections sous-jacentes) (10 %)[43,44] Diabète sucré[43,44] Troubles immunitaires (lupus érythémateux systémique, etc.)[43,44] Pathologies infectieuses (c.-à-d. VIH, hépatite, etc.)[43,44]	Sous-aiguë/ chronique	Symptômes généraux de maladie primaire ou secondaire : Malaises, gain pondéral, urine spumeuse, œdème périorbital du matin, ongles blanchâtres avec ou sans bandes blanches (lignes de Muehrcke), xanthomes cutanés et alopécie[42] Œdème périphérique retenant le godet marqué en soirée [31,42] Jaunisse[51] Perte de poids, polyurie, polydipsie, polyphagie Lupus érythémateux systémique : Arthrite et/ou arthralgie (86 %), éruptions cutanées (61 %), anémie (55 %), photosensibilité (48 %), fièvre (43 %), ulcère de la bouche (43 %), maux de tête (36 %), fatigue, malaise et faiblesse (35 %) et alopécie (35 %)[45]. Œdème généralisé ne retenant pas le godet[18] Œdème déclive retenant le godet à la fin du jour[18]	Analyse d'urine avec rapport protéines/créatinine, hémogramme et tests de coagulation, électrolytes pour vérifier la fonction rénale, tests de la fonction hépatique, glucose et HbA1C, tests ciblés pour les troubles décelés avec les antécédents et l'examen physique [43] anticorps antinucléaires, anticorps anti-ADN bicaténaires, anticorps anti-Sm, anticorps des anti-phospholipides, anticorps anti-ARN[42,43]	Néphrologue; rhumatologue
Néphropathie	Sous-aiguë/ chronique	Malaise ou inconfort abdominal[46], masse abdominale[46] ou bosse dorsocervicale[47], jaunisse[51], gain pondéral[41], œdème du visage, œdème abdominal, œdème périphérique retenant le godet[52]	Tests de la fonction rénale (inclure l'AST, l'ALT, le rapport AST/ ALT, la phosphatase alcaline et la γ-glutamyltransférase, la bilirubine, l'albumine, DFG), échographie hépatique ou IRM[46]	Hépatologue

Tableau 5 : *Mécanisme d'action des troubles systémiques accompagnant la chimiose conjonctivale*[2,6,18,33,40,42,49,53-58]

Étiologie			Mécanisme d'action Systémique
Atteintes du sinus caverneux	Fistule carotido-caverneuse		Altération du drainage veineux secondaire à une communication anormale entre l'artère carotide interne et le sinus caverneux ou les ramifications vers les méninges de l'artère carotide interne ou de l'artère carotide externe et des sinus caverneux[57]
	Thrombose carotido-caverneuse		Altération du drainage veineux secondaire à une infection du nez, des sinus, des oreilles ou des dents[32,58]
Hypersensibilité/allergie			Réaction allergique aux squames animales, au pollen, aux aliments, aux médicaments, au venin ou aux drogues induite par la libération d'histamine, de sérotonine et de bradykinine, accompagnée d'une migration des cellules polymorphonucléaires, laquelle provoque la dilatation des artérioles, la hausse du gradient de pression entre les capillaires artériolaires et les veinules ainsi que l'extravasation du liquide intravasculaire et œdème de vasodilatation consécutif[2,18,33]
Hyperthyroïdie			Accumulation de glycosaminoglycanes dans les tissus conjonctifs, entraînant un œdème et une inflammation des muscles extraoculaires, du tissu conjonctif orbital et adipeux, l'augmentation du volume orbital et la diminution du drainage veineux et lymphatique[49,53]
Syndrome de la veine cave supérieure	Lymphome		Ganglion lymphatique hypertrophié comprimant la veine supérieure et causant ainsi une diminution du drainage veineux[40]
	Bronchogène. Carcinome (cancer du poumon à petites cellules ou non)		Compression massive de la veine cave supérieure causant une diminution du drainage veineux[40]
	Tumeur des cellules germinales (prostate, thymus, adénocarcinome)		Compression massive de la veine cave supérieure causant une diminution du drainage veineux[40]
	Dispositifs intravasculaires		Dispositif qui comprime la veine cave supérieure causant une diminution du drainage veineux[40]
	Médastinite fibreuse (lymphome; histoplasmose; tuberculose; anévrisme de l'aorte d'origine syphilitique)		Cicatrisation fibreuse des tissus qui comprime la veine cave supérieure causant une diminution du drainage veineux[40]
	Autre (hématome après réparation par dissection aortique; pseudotumeur; thrombose primaire de la VCS)		Compression de la veine cave supérieure causant une diminution du drainage veineux[40]
Hypervolémie	Défaillance cardiorespiratoire		Dilatation du système vasculaire oculaire causée par l'hypoxie, l'hypercapnie, qui entrave le retour veineux thoracique en raison d'une ventilation sous pression positive qui induit une distension veineuse jugulaire et une surcharge de liquides[56]
	Syndrome néphrotique	Néphropathie primaire	Faible taux de protéines dans le sang (hypoprotéinémie) causant une diminution de la pression osmotique colloïdale, laquelle altère le gradient osmotique entre le plasma et le liquide interstitiel, causant ainsi la rétention du sel et de l'eau ainsi que l'évacuation des liquides dans l'espace extracellulaire [6,42,54]
		Néphropathie secondaire, en particulier le lupus érythémateux systémique	Patients présentant des signes d'affection systémique : Faible taux de protéines dans le sang (hypoprotéinémie) causant une diminution de la pression osmotique colloïdale, laquelle altère le gradient osmotique entre le plasma et le liquide interstitiel, causant ainsi l'évacuation du liquide dans l'espace extracellulaire [6,54] Patients ne présentant aucun signe d'affection systémique : Œdème localisé à partir de complexes immunitaires, causant une augmentation de la présence de cellules polymorphonucléaires et une augmentation de l'œdème et de l'inflammation[54,55]
	Affections hépatiques		Faible taux d'albumine dans le sang (hypoalbuminémie) causant une diminution de la pression osmotique colloïdale, une augmentation de la pression oncotique et l'évacuation des liquides dans les tissus mous oculaires[6]

Table 6: *Cutaneous presentations in systemic diseases that may present with conjunctival chemosis*[18,31,33,42,45,48,49,51,52,68-70]

Trouble Systémique		Présentation Cutanée				
		Rash	Alopecia (Yes Or No)	Jaundice (Yes Or No)	Edema (None; Pitting; Non-Pitting)	
Hypersensibilité/ allergie	D'origine médicamenteuse	Urticaire (bosses rougeâtres ou plaques pâles accompagnées d'œdème) 66,7 %[33]	Non	Non	Aucun(e)	
	D'origine alimentaire	Urticaire (bosses rougeâtres ou plaques pâles accompagnées d'œdème; 86,9 %[33]	Non	Non	Aucun(e)	
Hyperthyroïdie/maladie de Graves		Myxœdème prétibial se présentant avec un érythème asymptomatique chaud, humide, doux, velouté et lisse présentant une éléphantiasis prononcée (1-5 %)[49,52]	Oui[10]	Non	Myxœdème ne retenant pas le godet[31,52]	
Syndrome de la veine cave supérieure	Tumeur, ganglion lymphatique, dommages aux vaisseaux lymphatiques, radiothérapie[51]	Non	Non	Yes, if lymphoma51	Ne gardant pas le godet[52]	
Syphilis (se manifestant dans les zones extragénitales)	Primaire	Chancre without basal induration with edges rising above the surrounding surface (2%)[69]	Yes	Yes[51]	Aucun(e)	
	Secondaire	Maculaire, populaire, papulosquameux, lésion pustulaire (4,76 % à 59 %), selon l'emplacement, avec une présentation plus élevée sur les semelles, le tronc, les bras, les paumes, les jambes[70]				
	Tertiaire	Lésions nécrotiques centrées formant une gomme (15 %)[48]			Retenant le godet, si le patient souffre d'insuffisance cardiaque[52]	
Hypervolémie	Insuffisance cardiaque congestive	Non	Non	Oui, en présence d'insuffisance cardiaque et rénale[51]	Retenant le godet[51]	
	Syndrome néphrotique	Néphropathie primaire	Ongles blanchâtres avec ou sans bandes blanches (lignes de Muehrcke) et xanthomes cutanés[42]	Oui[48]	Oui[51]	Œdème généralisé ne retenant pas le godet [18,52] ou œdème déclive retenant le godet à la fin du jour[18]
		Lupus érythémateux systémique	Ongles blanchâtres avec ou sans bandes blanches (lignes de Muehrcke) et xanthomes cutanés[42] Toute forme d'éruption cutanée (> 80 %)[68] Éruption en forme de papillon (61 %)[45]			
	Néphropathie		Non	Oui[51]	Retenant le godet[52]	

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Predicting the Risk for Angle Closure as Defined by the Shaffer System Using Anterior Segment Optical Coherence Tomography: A Simple Approach

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Abstract

PURPOSE
To propose a simple non-invasive method for screening patients at risk for angle closure using anterior segment OCT.

METHODS
Scans of nasal and temporal iridocorneal angles in glaucoma suspect patients were performed using OCT. Upon identifying Schwalbe's line, the integrated caliper tool was used to draw a line to the nearest point of the iris to produce a measure 'S-I'. Gonioscopy was performed and angles graded according to Shaffer's classification to assess the correlation between both methods.

RESULTS
Thirty-four images were available for analysis. Spearman correlation coefficients between S-I anf gonioscopy grades were 0.81 for nasal and 0.77 for temporal quadrants respectively. Intraobserver ICC calculations demonstrated excellent reproducibility (0.98 and 0.99 for nasal and temporal angles) and excellent interobserver correlation (0.94 and 0.93). The diagnostic cutoff value of S-I for occludable angles was established at 330mm.

CONCLUSION
S-I measurement strongly correlates with gonioscopy and may be a suitable alternative for evaluating risk for angle closure.

KEY WORDS:
anterior chamber angle imaging, ASOCT, glaucoma, laser peripheral iridotomy, optical coherence tomography

Approximately 15 million people worldwide currently suffer from primary angle closure glaucoma (PACG);[1] this disease constitutes the leading cause of medically and surgically irreversible blindness. This type of glaucoma is caused by the apposition of peripheral iris onto the trabecular meshwork, resulting in obstructing and impeding aqueous humor outflow through the anterior chamber angle. Pupillary block, which accounts for 90% of PACG cases,[2] is caused by apposition of the iris onto the anterior face of the lens, which restricts aqueous humor flow from the posterior to anterior chamber. The accumulation of aqueous humor in the posterior chamber causes anterior bowing of the iris and eventual angle closure. This mechanism can occur acutely, intermittently or chronically. The mean prevalence for PACG worldwide is currently estimated at 0.69%.[1]

In the last 40 years, numerous risk factors have been identified, such as hyperopic eyes, small corneas and high iris insertion, but observation of the irido-corneal angle through gonioscopy remains the clinical standard when screening for this disease. Although easily carried out, gonioscopy presents its limitations: the necessity for direct contact with an anesthetized cornea,

skewed results caused by pressure on the cornea or too much ambient light artificially opening the angle. Moreover, multiple studies have demonstrated great variability in gonioscopic findings among experienced observers.[3-5]

With the advent of optical coherence tomography (OCT) in ophthalmology,[6] its use was previously exclusive to the study of the posterior segment (retina). Nowadays rapid acquisition of high definition anterior segment images is possible, all the while eliminating the need for corneal contact.[7]

Multiple studies have already described different approaches for quantitatively defining the irido-corneal opening,[7-10] while using the scleral spur as their anatomical landmark. Being non-identifiable 20-30% of the time,[8] authors quickly turned towards Schwalbe's line as their new anatomical reference;[9-10] which can be visualized and identified in more than 95% of the anterior segment OCT images. Current models of measure compute anterior segment characteristics such as Angle Opening Distance (AOD), Trabecular-Iris-Surface-Area (TISA) and Schwalbe's line Angle Opening Distance (SL-AOD) which have been shown to have good inter and intraobserver reliability. However, these measurements still rely on observer caliper manipulations on several image points which are potentially subject to manipulation errors. Although some OCT instruments can readily compute these measurements, many other devices cannot, requiring exporting the images onto another platform, increasing the time needed for image analysis.

In this study, we suggest a simple approach using anterior segment optical coherence tomography, or AS-OCT imaging, which would allow one to rapidly image and quantify the irido-corneal opening by determining the minimal distance between Schwalbe's line and iris. A single line traced from iris to Schwalbe's line could potentially be simpler to draw than exporting an image and placing a multi-point caliper on the anterior segment angle. This tool could be used as a quicker and non-invasive screening method, for patients in which angle closure is suspected.

METHODS

Subjects

In this prospective study, forty Caucasian subjects (22 females, 18 males) were recruited from the glaucoma clinic at the **Institut de l'Oeil des Laurentides (IOL)**, in Boisbriand (Quebec). The patients were seen for the first time at the Institute and had been referred by their respective optometrist as glaucoma suspects, whether because they had elevated intra-ocular pressure, suspicious cup-to-disc ratio, etc. Written informed consent was obtained from all participants, after the study had received approval from Montreal University's ethics committee review board (Certificate #13-029-CERES-D), in accordance to the tenets of the Declaration of Helsinki.

All subjects included underwent ophthalmic examination, including visual acuity, slit-lamp biomiscroscopy, Goldmann applanation tonometry and darkroom gonioscopy. Subjects with a history of previous intraocular surgery, penetrating trauma, pigment dispersion syndrome or any corneal opacities or abnormalities that precluded AS-OCT imaging were excluded. Those having previously undergone laser peripheral iridotomy (LPI) were not excluded.

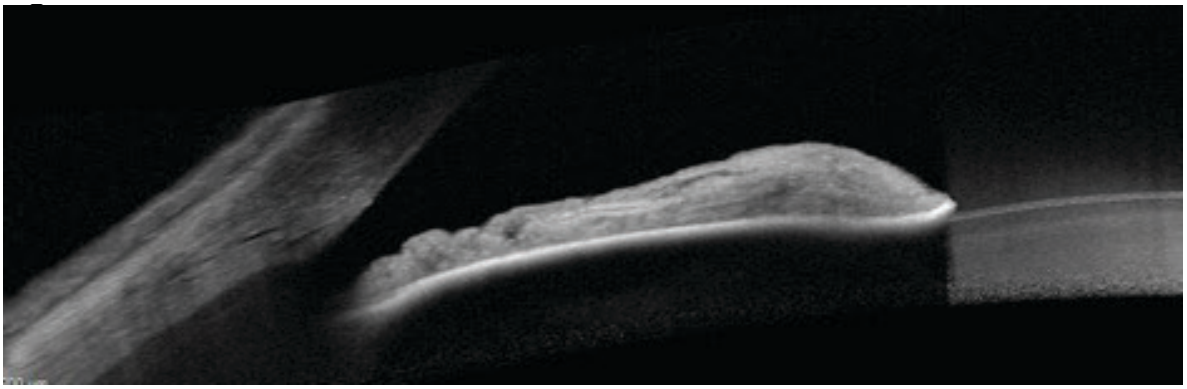
An experienced glaucoma specialist (SG) performed all the aforementioned exams, and used a G4 Volk 4-mirror gonioscope in a darkened examination room. Using a 1 mm wide and 7 mm high slit beam, vertically offsetting the light to enable corneal wedge visualization and avoiding light exposure through the pupil, grading was recorded for 4 angle quadrants (superior, inferior, nasal and temporal) in both eyes using the Shaffer grade classification. According to the American Academy of Ophthalmology (AAO) this classification is the most commonly used. Grading properties were as follows: grade 4 for a wide-open angle (35-45 degrees); grade 3 for a wide-open angle (20-35 degrees); grade 2 was used to denote an angle with a possible risk of closure (20 degrees); a quadrant was graded at 1 or less (10 degrees or less) to signify a high risk of closure. Indentation gonioscopy was performed to verify the presence or absence of peripheral anterior synechiae (PAS). Patients with evidence of PAS were excluded.

ANTERIOR CHAMBER AND ANGLE IMAGING

AS OCT

Imaging of the angle was performed on undilated pupils, with a Fourier-domain (spectral) high definition optical coherence or SD-OCT system (Spectralis, Heidelberg Engineering, Germany). The apparatus utilizes an 820nm wavelength light source and has a scanning speed of 40,000 A-scans/second. Image lateral resolution can reach 7 microns per pixel. An anterior segment objective lens was mounted for anterior segment imaging. All scans were performed under uniform dim illumination of 3.5 lux, as measured with a luxmeter (Sekonic model L-308DC, Digi-CineMate, North White Plains, NY). Scans were acquired with the 'angle to angle' module to image both nasal and temporal angles at the same time (**Figure 1**), on the same image. A fixation target was utilized to properly guide the gaze of the subject into primary position. Only images of nasal and temporal quadrants were acquired, due to technical difficulties in scan acquisition of the superior and inferior quadrants.

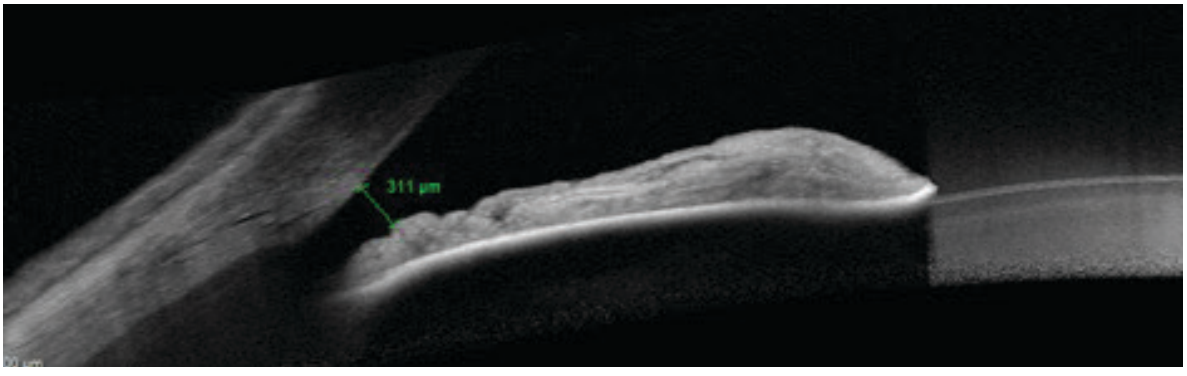
Figure 1: *Anterior segment Imaging using Spectralis optical coherence tomography; nasal and temporal angles in a single capture.*



Quantitative assessment: S-I

Images presenting artifacts hindering identification of anatomical landmarks (Schwalbe's line) were excluded. Two observers (DS, SG) independently carried out the measurements. After identifying Schwalbe's line, a line was drawn between S and the closest point of the iris using the Spectralis line tracer integrated tool. The measurement was instantaneously displayed in microns and the observer could therefore readily identify the smallest measure possible. We called this measure S-I (**Figure 2).** Observer DS re-graded the randomly reordered images two weeks later.

Figure 2: *Anterior chamber angle in an anterior segment optical coherence tomography line scan image. (A) One can clearly identify Schwalbe's line as the little indentation signifying the termination of the corneal endothelium. (B) S-I measurement using the integrated measurement tool. The distance is automatically displayed and shown here as 311 μm.*



Statistical analysis

One eye from each subject was randomly selected for data analysis. Statistical analyses were performed using SPSS version 25 (developed by SPSS, Chicago, IL). The chi-square test was used to compare categorical data. ANOVA and Tukey Kramer tests were used to assess the difference between S-I means for each gonioscopic grade to evaluate whether their differences were significant, S-I being quadrant-specific (nasal and temporal). An intraclass coefficient correlation analysis (ICC) was used to assess inter and intraobserver reproducibility. Finally, Spearman's Rho coefficient was used to evaluate the strength of association between S-I and gonioscopic grading.

RESULTS

All patients were Caucasian. The mean age (Standard Deviation SD) was 59.7 years (7.67, range 41-75) and average spherical equivalent was +0.75 ± 1.50 D. Eight images were excluded owing to poor quality, leaving 32 images available for analysis (14 right eyes and 18 left eyes). There were no significant differences in mean age and gender for all included subjects (using Chi –square analysis, data not shown). Seventeen patients were found to have gonioscopically occludable angles (Shaffer grade 1 or less), five of which had already previously undergone peripheral laser iridotomy. The correlation between S-I measurements and gonioscopic grading was found to be statistically significant (Spearman r = 0.81 and 0.79 for the nasal and temporal quadrants respectively, $\boldsymbol{p}$<0.05).

Table 1 demonstrates results from Tukey Kramer HSD testing, illustrating the differences between means of S-I for each gonioscopic grade for nasal and temporal quadrants respectively. Positive values indicate a pairing that is significantly different. For example, looking at table 1, the first line compares the mean S-I value for a Shaffer grade 4 with the mean S-I value of all the other Shaffer grades. One can observe that the mean S-I value for a grade 4 is significantly different from S-I means from Shaffer grades 2, 1 and 0. Figure 3 graphically represents those results, accompanied by the comparison circles.

Table 1: *Comparison between means using Tukey-Kramer HSD test (OCT S-I N vs Gonio grade)*

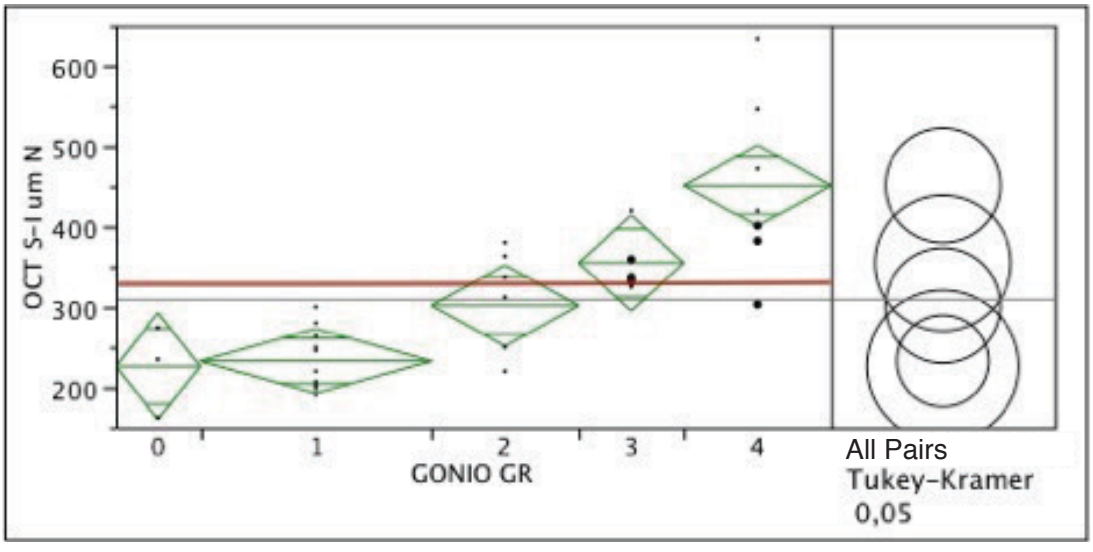
SHAFFER GRADE	4	3	2	1	0
4	X	-14.1978	28.342	126.3621	106.3621
3	-14.1978	x	5.988	19.90661	1.64431
2	28.3242	5.988	x	-3.427	-9.621
1	126.8587	19.90661	-3.427	x	-103.715
0	106.3621	1.64431	-9.621	-103.715	x

*Positive values show pairs of means that are statistically different

GRADE		AVERAGE
4	A	451.00
3	A B	354.60
2	B C	301.71
1	C	232.81
0	C	226.25

Levels not connected by the same letter are largely different

Figure 3: *Comparison of OCT S-I Nasal means with gonioscopic grading using Tukey-Kramer HSD analysis. The Younen index was used to determine the cutoff value for S-I between open angles and occludable angles (red line).*



Interobserver ICC calculations demonstrated excellent reproducibility using ICC analysis (0.94 and 0.93, $p<0.001$ for nasal and temporal quadrants respectively). Using the same method, the analysis also revealed excellent intraobserver reproducibility (0.98 and 0.99, $p<0.001$ for nasal and temporal quadrants respectively).

DISCUSSION

This study aimed to devise a new, simple parameter to quantify the risk of angle closure, using Schwalbe's line as identified on anterior segment OCT imaging. Devoid of the need to transfer images on some of the available OCT instruments, use complex mathematical logarithms, or manipulate several caliper points on acquired images, our results demonstrated an excellent correlation between our S-I measurement and standard gonioscopic grading. This simple quantitative measure can be used to rapidly screen and monitor patients in whom angle closure is suspected while removing the necessity to topically anesthetize and create contact with the cornea of the patient. Moreover, this technique requires less operator manipulations to compute when compared with methods such as TISA and AOD measurements and eliminates the need for image transferring.

With the advent of high resolution imaging of the anterior chamber angle, visualization of SL is now more readily achieved; it was identified in 94% of analyzable images, a finding similar to previous studies.[10,15] Other researchers have used the Sclera Spur or Trabecular Meshwork as the anatomical landmarks for the measurement of their parameters.[16-17] A major limitation of using such landmarks is that they are only identifiable on 70-80% of HD-OCT images. Thus, our findings showed that our parameter using SL as the anatomical landmark may be more suitable in assessing irido-corneal angles with respect to OCT imaging.

Figure 3 is a graphic illustration of the Tukey-Kramer HSD test results for the nasal quadrant. The results are fairly identical to the temporal quadrant (results not shown). The comparison circles of S-I values corresponding to gonioscopic grades 3 and 4 are significantly separated from S-I values corresponding to gonioscopically occludable angles. The red line displays the cutoff value for S-I (330mm) between wide-open angles and those at risk for closure, as defined by ROC analysis and subsequent identification of Younen's index (table not shown).

One of the main limitations of this study is the lack of acquisition of HD-OCT images for the superior and inferior quadrants, owing to technical difficulties (eyelids and the need for a speculum). It is possible that the pattern of visibility of anatomical landmarks in those quadrants is different. Secondly, with HD-OCT imaging, an external fixation light must be used to properly position the eye of the subject and image the horizontal quadrants. The fixation pattern or position of the fixation light have yet to be standardized. Furthermore, all subjects in the study were Caucasian; as angle closure is more prevalent in other ethnicities, such as Asians, African-Americans and Eskimos, repeating the study on such groups might prove very useful to further characterize S-I across different populations. Finally, the relatively small sample size poses as another potential limitation.

CONCLUSION

Primary angle closure glaucoma is a sight-threatening disease. Current methods of screening include gonioscopy and UBM, both of which make surface contact with the eye and are difficult to master. Anterior segment imaging using HD-OCT is a promising and non-invasive method, but remains unstandardized in the screening of occludable angles. Results using our novel, and for some users, simpler measurement parameter, suggest S-I may be potentially useful in rapidly and easily quantifying the risk for angle closure. A cutoff value between open and closed irido-corneal angle was also provisionally provided (330 mm). Further studies should be elaborated to answer the aforementioned limitations. ●

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RECHERCHE CLINIQUE

Prévoir le risque de fermeture de l'angle tel que défini par la classification de Shaffer à l'aide de la tomographie par cohérence optique du segment antérieur : Une approche simple

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OBJECTIF
Proposer une méthode non invasive simple pour le dépistage des patients à risque de fermeture de l'angle en utilisant l'imagerie du segment antérieur à l'aide de la TCO.

MÉTHODES
Des examens radiologiques des angles irido-cornéens en nasal et en temporal ont été réalisés au moyen de la TCO chez des patients référés pour suspicion de glaucome. Après l'identification de la ligne de Schwalbe, une droite fut tracée jusqu'au point de l'iris le plus rapproché (S-I) à l'aide de l'outil de compas intégré de l'appareil. Une gonioscopie a été effectuée et la méthode de Shaffer a été utilisée pour grader l'ouverture de l'angle afin d'évaluer la corrélation entre les deux méthodes.

RÉSULTATS
Trente-quatre images étaient disponibles pour analyse. Les coefficients de corrélation de Spearman entre les mesures S-I et les grades de Shaffer se sont établis à 0,81 et 0,77 pour les quadrants nasal et temporal respectivement. Les calculs intra-observateur des coefficients de corrélation intra-classe avaient une excellente reproductibilité (0,98 et 0,99 pour les angles en nasal et en temporal) et on a observé une excellente corrélation inter-observateur (0,94 et 0,93). La valeur diagnostique S-I pour les angles à risque de fermeture a été provisoirement établie à 330 mm.

CONCLUSION
La mesure S-I corrèle fortement avec les résultats de la gonioscopie et pourrait être une solution de rechange appropriée pour évaluer le risque de fermeture de l'angle.

MOTS CLÉS :
imagerie de l'angle de la chambre antérieure, TCO-SA, glaucome, iridotomie périphérique au laser, tomographie par cohérence optique

À l'heure actuelle, environ 15 millions de personnes dans le monde souffrent de glaucome primitif à angle fermé (GPAF)[1]; cette maladie constitue la première cause de cécité irréversible médicalement ou chirurgicalement. Ce type de glaucome est causé par l'accolement de la partie périphérique de l'iris sur le réseau trabéculaire, ce qui empêche l'écoulement de l'humeur aqueuse par l'angle de la chambre antérieure. Le bloc pupillaire, qui est à l'origine de 90 % des cas de GPAF[2], est causé par un accolement de l'iris et de la face antérieure du cristallin, qui limite l'écoulement de l'humeur aqueuse de la chambre postérieure vers la chambre antérieure. L'accumulation de l'humeur aqueuse dans la chambre postérieure pousse l'iris de façon périphérique vers l'avant (iris bombé) et, éventuellement, cause la fermeture de l'angle. Ce mécanisme peut se produire de façon aiguë, intermittente ou chronique. La prévalence moyenne du GPAF dans le monde est actuellement estimée à 0,69 %[1].

Au cours des 40 dernières années, de nombreux facteurs de risque ont été identifiés, comme l'hypermétropie, un faible diamètre cornéen et l'insertion élevée de l'iris, mais l'observation de l'angle irido-cornéen par gonioscopie demeure la norme clinique pour le dépistage de cette maladie. Bien qu'elle soit facile à réaliser, la gonioscopie présente ses limites, à savoir la nécessité d'un contact direct avec une cornée anesthésiée, les résultats faussés en raison de la pression sur la cornée ou d'une trop grande quantité de lumière ambiante qui provoque une ouverture artificielle de l'angle. De plus, de nombreuses études ont démontré la grande variabilité des résultats obtenus par gonioscopie chez les observateurs expérimentés[3-5].

L'utilisation de la tomographie par cohérence optique (TCO) en ophtalmologie était auparavant réservée à l'étude du segment postérieur (rétine)[6]. De nos jours, il est possible d'acquérir rapidement des images en haute définition du segment antérieur, tout en éliminant le besoin de contact avec la cornée[7].

De nombreuses études ont déjà décrit différentes approches pour définir quantitativement l'ouverture de l'angle irido-cornéen[7-10] en utilisant l'éperon scléral comme point de repère anatomique. Comme il n'est pas identifiable dans 20 à 30 % des cas[8], les auteurs se sont rapidement tournés vers la ligne de Schwalbe comme nouveau point de repère anatomique[9-10], puisqu'elle peut être visualisée et identifiée sur plus de 95 % des images du segment antérieur acquises par TCO. Les modèles actuels de mesure calculent les caractéristiques des segments antérieurs, comme la distance d'ouverture de l'angle (AOD), la surface de l'iris trabéculaire (TISA) et la distance d'ouverture de l'angle de la ligne de Schwalbe (SL-AOD) dont la fiabilité inter-observateur et intra-observateur a été démontrée. Toutefois, ces mesures reposent toujours sur des manipulations de compas par les observateurs à plusieurs points d'image et peuvent faire l'objet d'erreurs de manipulation. Bien que certains instruments de TCO puissent facilement calculer ces mesures, beaucoup d'autres dispositifs ne le peuvent pas. Il est donc nécessaire d'exporter les images sur une autre plateforme, ce qui augmente le temps nécessaire à l'analyse des images.

Dans cette étude, nous proposons une approche simple utilisant la tomographie par cohérence optique du segment antérieur, ou TCO-SA, qui permettrait d'obtenir des images et de quantifier l'ouverture de l'angle irido-cornéen rapidement en déterminant la distance minimale entre la ligne de Schwalbe et l'iris. Il pourrait être plus simple de tracer une seule ligne de l'iris à la ligne de Schwalbe que d'exporter une image et d'utiliser un outil de compas à plusieurs points de l'angle du segment antérieur. Cet outil pourrait être utilisé comme méthode de dépistage plus rapide et non invasive pour les patients chez qui on soupçonne la fermeture de l'angle.

MÉTHODES

Participants

Dans cette étude prospective, 40 sujets blancs (22 femmes, 18 hommes) ont été recrutés à la clinique de glaucome de l'**Institut de l'Œil des Laurentides (IOL)** située à Boisbriand (Québec). Les patients ont été vus pour la première fois à l'Institut et avaient été référés par leur optométriste respectif pour suspicion de glaucome, en raison, notamment, d'une pression intraoculaire élevée ou d'un rapport cupule-disque suspect., L'étude a reçu l'approbation du comité d'éthique de l'Université de Montréal (certificat #13-029-CERES-D) et le consentement éclairé par écrit de tous les participants a été obtenu, conformément aux principes de la Déclaration d'Helsinki.

Tous les sujets ont subi un examen ophtalmique comprenant l'acuité visuelle, la biomiscroscopie par lampe à fente, la tonométrie par applanation (tonomètre de Goldmann) et la gonioscopie en chambre noire. Les sujets ayant des antécédents de chirurgie intraoculaire, de traumatisme pénétrant, de syndrome de dispersion des pigments ou toute opacité ou anomalie cornéenne en raison de laquelle il n'était pas possible de réaliser une TCO-SA ont été exclus. Les personnes ayant déjà subi une iridotomie périphérique au laser n'ont pas été exclues.

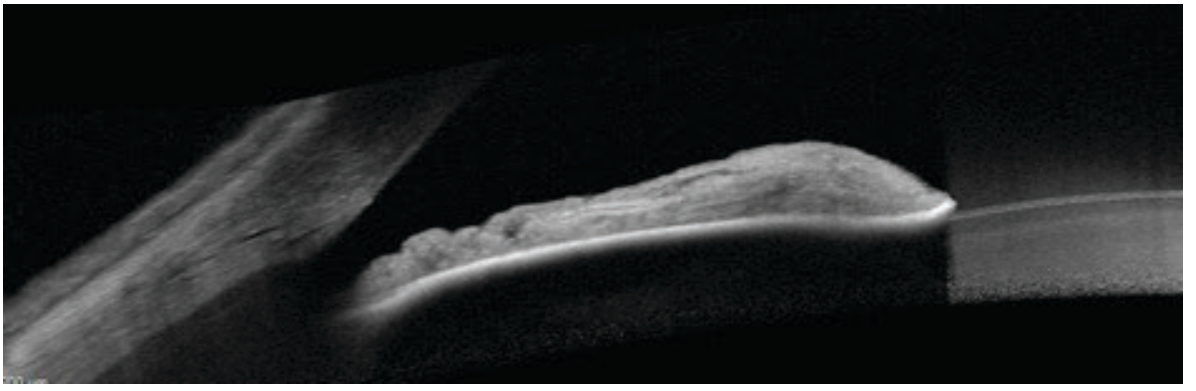
Un spécialiste du glaucome expérimenté (SG) a effectué tous les examens susmentionnés et a utilisé un gonioscope à 4 miroirs G4 Volk dans une salle d'examen sombre. À l'aide d'un faisceau à fente de 1 mm de largeur et de 7 mm de hauteur, en décalant verticalement la lumière pour permettre la visualisation de l'angle cornéen et éviter l'exposition à la lumière par la pupille, on a gradé l'angle dans les quatre quadrants (supérieur, inférieur, nasal et temporal) des deux yeux en utilisant la classification de Shaffer. Selon l'American Academy of Ophthalmology (AAO), cette classification est la plus couramment utilisée. La classification de Shaffer est la suivante : niveau 4 pour un angle ouvert (35-45 degrés); niveau 3 pour un angle ouvert (20-35 degrés); niveau 2 pour indiquer un angle avec un risque de fermeture (20 degrés); niveau 1 ou moins pour un quadrant (10 degrés ou moins) pour indiquer un risque élevé de fermeture. On a procédé à une gonioscopie par indentation pour vérifier la présence ou l'absence de synéchie antérieure périphérique (SAP). Les patients présentant des signes de SAP ont été exclus.

IMAGERIE DE LA CHAMBRE ANTÉRIEURE ET DE L'ANGLE

TCO-SA

L'imagerie de l'angle a été réalisée sur des pupilles non dilatées, avec un système de tomographie par cohérence optique en domaine spectral (TCO dans le domaine de Fourier) ou TCO-DS (Spectralis, Heidelberg Engineering, Allemagne). L'appareil utilise une source de lumière d'une longueur d'onde de 820 nm et a une vitesse de balayage de 40 000 A-scans par seconde. La résolution latérale de l'image peut atteindre 7 microns par pixel. Un objectif pour segment antérieur a été utilisé pour l'imagerie du segment antérieur. Tous les balayages ont été effectués sous un éclairage uniforme de 3,5 lux, mesuré à l'aide d'un luxmètre (modèle Sekonic L-308DC, DigiCineMate, North White Plains, NY). Des balayages ont été effectués au moyen du module « angle-angle » pour visualiser les angles en nasal et en temporal simultanément (**figure 1**) sur la même image. Une cible de fixation a été utilisée pour guider correctement le regard du sujet vers sa position principale. Seules des images de quadrants nasal et temporal ont été acquises, en raison de difficultés techniques dans l'acquisition par balayage des quadrants supérieur et inférieur.

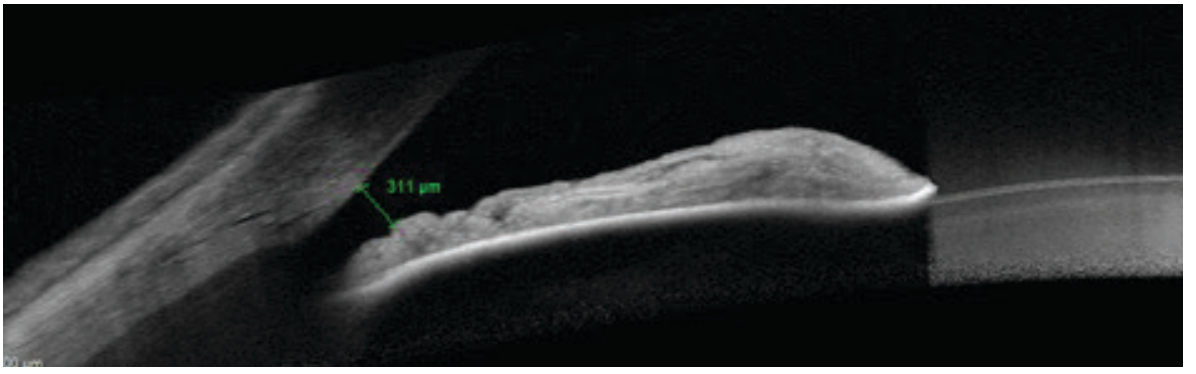
Figure 1 : *Imagerie du segment antérieur à l'aide de la tomographie par cohérence optique Spectralis; angles nasal et temporal en une seule capture.*



Évaluation quantitative : S-I

Les images présentant des artefacts qui entravaient l'identification du point de repère anatomique (ligne de Schwalbe) ont été exclues. Deux observateurs (DS, SG) ont effectué les mesures de façon indépendante. Après avoir identifié la ligne de Schwalbe, une ligne a été tracée entre celle-ci et le point le plus proche de l'iris à l'aide de l'outil intégré de traceur de ligne du Spectralis. La mesure était instantanément affichée en microns et l'observateur pouvait donc facilement identifier la plus petite mesure possible. Nous avons appelé cette mesure S-I (**figure 2).** L'observateur DS a réévalué les images réorganisées de façon aléatoire deux semaines plus tard.

Figure 2 : *Angle de la chambre antérieure dans une image par tomographie par cohérence optique du segment antérieur. (A) On peut clairement identifier la ligne de Schwalbe comme la petite indentation signifiant la fin de l'endothélium cornéen. (B) Mesure S-I à l'aide de l'outil de mesure intégré. La distance est affichée automatiquement et indiquée ici comme étant 311* µm.



Analyse statistique

Pour chaque sujet, un œil a été sélectionné au hasard pour l'analyse des données. Les analyses statistiques ont été effectuées à l'aide de la version 25 de SPSS (élaborée par SPSS, Chicago, IL). Le test du chi carré a été utilisé pour comparer des données catégorielles. Pour chaque grade par gonioscopie, les tests ANOVA et Tukey Kramer ont été utilisés pour analyser les paires de moyenne de la valeur S-I et évaluer si la différence était significative, la mesure S-I étant spécifique au quadrant (nasal et temporal). Une analyse de corrélation des coefficients intraclasse (CCI) a été utilisée pour évaluer la reproductibilité inter-observateur et intra-observateur. Finalement, le coefficient rhô de Spearman a été utilisé pour évaluer la force de l'association entre la valeur S-I et le classement par gonioscopie.

RÉSULTATS

Tous les patients étaient de race blanche. L'âge moyen (écart type ET) était de 59,7 ans (7,67, plage 41-75) et l'équivalent sphérique moyen était de +0,75 1,50 D. Huit images ont été exclues en raison de leur piètre qualité, ce qui a laissé 32 images à analyser (14 yeux de droite et 18 yeux de gauche). Il n'y avait pas de différences significatives entre l'âge moyen et le sexe pour tous les sujets inclus (analyse chi carré, données non présentées). Dix-sept patients présentaient des angles à risque de fermeture par gonioscopie (grade Shaffer 1 ou moins), dont cinq qui avaient déjà subi une iridotomie périphérique au laser. La corrélation entre les mesures S-I et le classement par gonioscopie a été jugée statistiquement significative (Spearman r = 0,81 et 0,79 pour les quadrants nasal et temporal respectivement, $p < 0,05$).

Le **tableau 1** présente les résultats du test DSH de Tukey Kramer et illustre les différences entre les moyennes S-I pour chaque grade gonioscopique pour les quadrants nasal et temporal, respectivement. Les valeurs positives indiquent une paire d'angle dont la valeur diffère de façon statistiquement significative. Par exemple, en regardant le tableau 1, la première ligne compare la valeur moyenne S-I pour un grade 4 de Shaffer avec la valeur moyenne S-I pour tous les autres grades de Shaffer. On peut observer que la valeur moyenne S-I pour un grade 4 est très différente de la valeur S-I pour un grade 2, 1 ou 0 de Shaffer. La figure 3 illustre graphiquement ces résultats, accompagnés des cercles de comparaison.

Tableau 1 : *Comparaison entre les moyennes à l'aide du test DSH de Tukey-Kramer (valeur S-I obtenue par TCO par rapport au grade défini par gonioscopie)*

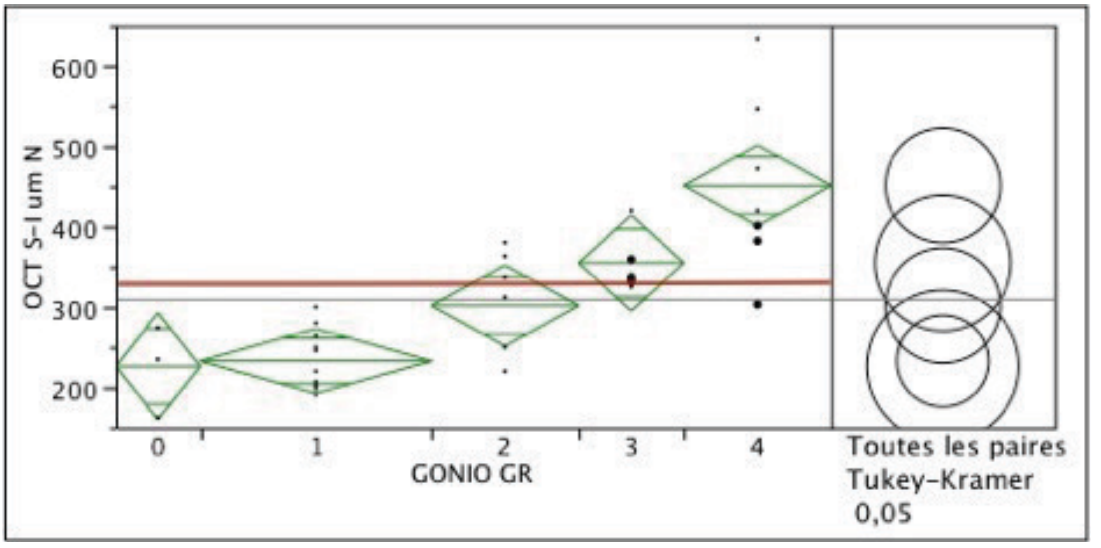
Grade SHAFFER	4	3	2	1	0
4	X	-14,1978	28,342	126,3621	106,3621
3	-14,1978	x	5,988	19,90661	1,64431
2	28,3242	5,988	x	-3,427	-9,621
1	126,8587	19,90661	-3,427	x	-103,715
0	106,3621	1,64431	-9,621	-103,715	x

*Les valeurs positives révèlent des paires de moyennes qui sont statistiquement différentes

GRADE		MOYENNE
4	A	451,00
3	A B	354,60
2	B C	301,71
1	C	232,81
0	C	226,25

Les niveaux qui ne sont pas reliés par la même lettre présentent une différence importante

Figure 3 : *Comparaison des moyennes de la mesure S-I en nasal par TCO avec le classement par gonioscopie à l'aide de l'analyse DSH de Tukey-Kramer. L'indice Younen a été utilisé pour déterminer la valeur seuil S-I entre les angles ouverts et les angles à risque de fermeture (ligne rouge).*



L'analyse du CCI a montré que les calculs du CCI inter-observateur avaient une excellente reproductibilité (0,94 et 0,93, $p < 0{,}001$ pour les quadrants nasal et temporal respectivement). En utilisant la même méthode, l'analyse a également révélé une excellente reproductibilité intra-observateur (0,98 et 0,99, $p < 0{,}001$ pour les quadrants nasal et temporal respectivement).

DISCUSSION

Cette étude visait à concevoir un nouveau paramètre simple pour quantifier le risque de fermeture de l'angle, en utilisant la ligne de Schwalbe identifiée sur l'image par TCO du segment antérieur. Éliminant la nécessité de transférer des images sur certains des instruments de TCO disponibles, d'utiliser des logarithmes mathématiques complexes ou de manipuler plusieurs points de compas sur des images acquises, nos résultats ont démontré une excellente corrélation entre nos mesures S-I et le classement par gonioscopie standard. Cette mesure quantitative simple peut être utilisée pour faire un dépistage rapidement chez les patients chez lesquels on soupçonne une fermeture de l'angle tout en éliminant la nécessité d'anesthésier par voie topique et de créer un contact avec la cornée du patient. De plus, cette technique nécessite moins de manipulations de l'opérateur pour effectuer des calculs par rapport à des méthodes comme les mesures TISA et AOD et élimine le besoin de transfert d'images.

Avec l'avènement de l'imagerie haute résolution de l'angle de la chambre antérieure, la visualisation de la ligne de Schwalbe est désormais plus facile ; elle a été identifiée dans 94 % des images analysables, un résultat semblable à ceux des études précédentes[10,15]. D'autres chercheurs ont utilisé l'éperon scléral ou le réseau trabéculaire comme repères anatomiques pour la mesure de leurs paramètres[16-17]. Une limitation majeure de l'utilisation de ces repères est qu'ils ne sont identifiables que sur 70 à 80 % des images TCO-HD. Ainsi, nos résultats ont montré que notre paramètre utilisant la ligne de Schwalbe comme point de repère anatomique peut être plus approprié que la TCO pour évaluer les angles irido-cornéens.

La figure 3 est une illustration graphique des résultats du test DSH de Tukey-Kramer pour le quadrant nasal. Les résultats sont semblables à ceux obtenus pour le quadrant temporal (résultats non présentés). Les cercles de comparaison des valeurs S-I correspondant aux grades 3 et 4 établis par gonioscopie et des valeurs S-I correspondant aux angles à risque de fermeture d'après la gonioscopie montrent une différence statistiquement significative. La ligne rouge affiche la valeur seuil S-I (330 mm) entre les angles ouverts et ceux à risque de fermeture, telle que définie par l'analyse ROC et l'identification subséquente de l'indice de Younen (tableau non présenté).

L'une des principales limites de cette étude est le manque d'acquisition d'images TCO-HD pour les quadrants supérieur et inférieur, en raison de difficultés techniques (paupières et besoin d'un speculum). Il est possible que le

schéma de visibilité des repères anatomiques dans ces quadrants soit différent. Deuxièmement, avec l'imagerie TCO-HD, une lampe de fixation externe doit être utilisée pour bien positionner l'œil du sujet et visualiser les quadrants horizontaux. La position de fixation de la lampe de fixation n'a pas encore été normalisée. De plus, tous les sujets de l'étude étaient de race blanche. La fermeture de l'angle étant plus répandue dans d'autres ethnies, comme les Asiatiques, les Afro-Américains et les Inuits, il pourrait être très utile de répéter l'étude sur ces groupes pour mieux caractériser S-I dans différentes populations. Enfin, la taille relativement petite de l'échantillon constitue une autre limite potentielle.

CONCLUSION

Le glaucome primitif à angle fermé est une maladie qui peut causer la cécité. Les méthodes actuelles de dépistage comprennent la gonioscopie et l'UBM, qui exigent toutes deux le contact de surface avec l'œil et sont difficiles à maîtriser. L'imagerie du segment antérieur par TCO-HD est une méthode prometteuse et non invasive, mais son utilisation n'est pas normalisée pour le dépistage des angles à risque de fermeture. Les résultats obtenus en utilisant notre nouveau paramètre de mesure, méthode qui peut s'avérer plus simple pour certains utilisateurs, montrent que S-I pourrait être utile pour quantifier rapidement et facilement le risque de fermeture de l'angle. Une valeur seuil entre l'angle irido-cornéen ouvert et fermé a également été fournie provisoirement (330 mm). D'autres études devront être menées pour répondre aux limites susmentionnées. ●

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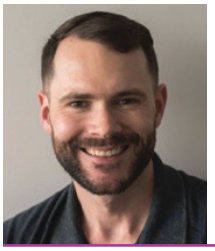
Why Optometrists Need to be Blogging



Cameron Martel
Cameron Martel is an experienced digital marketer, managing SEO and content campaigns since 2005. He currently works with dozens of eye care practices through his work with Marketing4ECPs. He's colourblind, but don't remind him, or he'll be seeing red (or so he thinks). Cam can be reached at www.marketing4ecps.com or cameron@4ecps.com.

We are huge proponents of blogging for Optometry practices. It has proven to be one of the most effective ways to generate new traffic, increase brand awareness, and attract new patients. That's why we have made it one of the pillars of our SEO (search engine optimization) strategy.

Blogging has fallen out of fashion, and those that do it often don't do it well. Many practices are focusing more on social media, leaving their blog rarely - if ever - updated. Let's explore why that's a missed opportunity for them, and a massive one for you.

WHY BLOGGING IS ONE OF THE MOST POWERFUL ORGANIC MARKETING TOOLS AVAILABLE

Google loves great content. It loves when its users find themselves down the deep-dive rabbit hole, exploring blog after blog and consuming copious amounts of information.

Why?

Because that means its users are finding value in the results Google shows, and if that's true, it means that those users will come back again and again. With more than 90% of the search market share, it stands to reason that their logic rings true.

It's also one of the least expensive ways to generate leads. If you create the blog yourself, all it costs is a small portion of your time. When done correctly, blogging can transform a business. Really.

THE OPPORTUNITY BLOGGING REPRESENTS

Those that scoff at blogging ignore two important points: first, that users *want* this information and actively seek it out; second, Google *wants* to serve this information from *subject matter experts* like you. In fact, in August of 2018, Google made a significant change to its algorithm that favours medical professionals for medically-focused keywords. In other words, when it comes to keywords surrounding eye care, Optometrists have a significant leg-up.

From a business point of view, blogging has four major benefits:

Educate & Qualify Future Patients & Customers
How many times have you had to defend the price of an eye exam or a pair of frames? The reality is that most people simply don't know what they don't know. Via your blog, you have a way to educate people about eye health, vision correction, eyewear, and what goes into them.

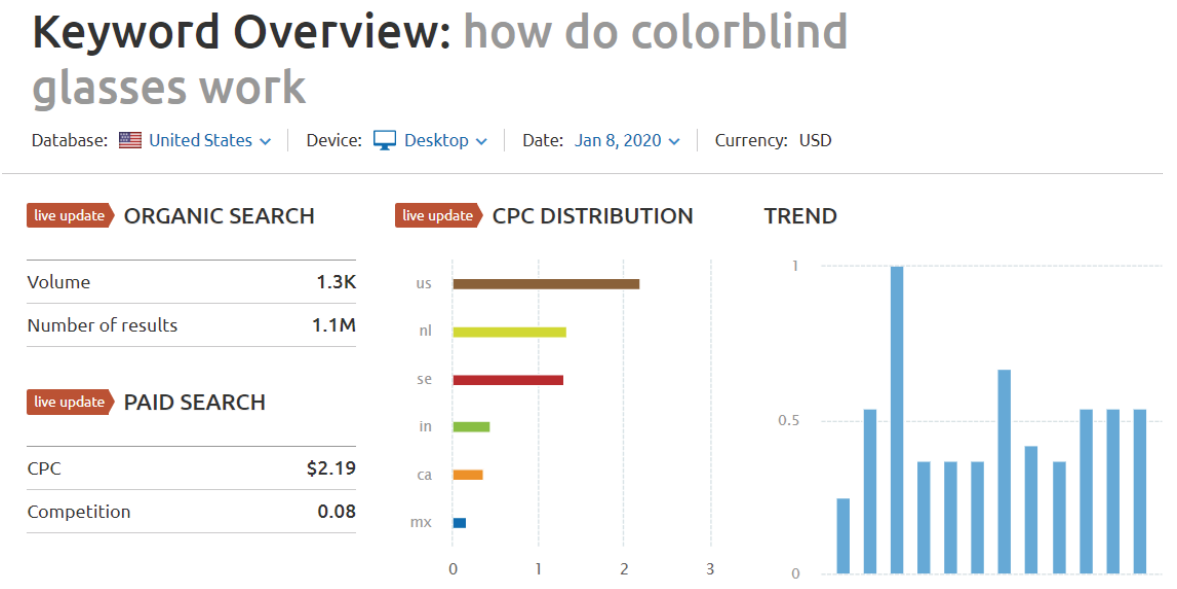
An educated patient is more likely to see your value and far less likely to shop based solely on price.

Demonstrate Expertise & Thought Leadership
In an era of fake news and misinformation, people seek information from experts they can genuinely trust. If you can teach them about something they care about, you will earn their trust.

The content you create for your blog can be easily shared on your social channels, including LinkedIn. By helping people answer their questions, you earn their trust and advocacy. That's powerful and not something you can accomplish via traditional advertising.

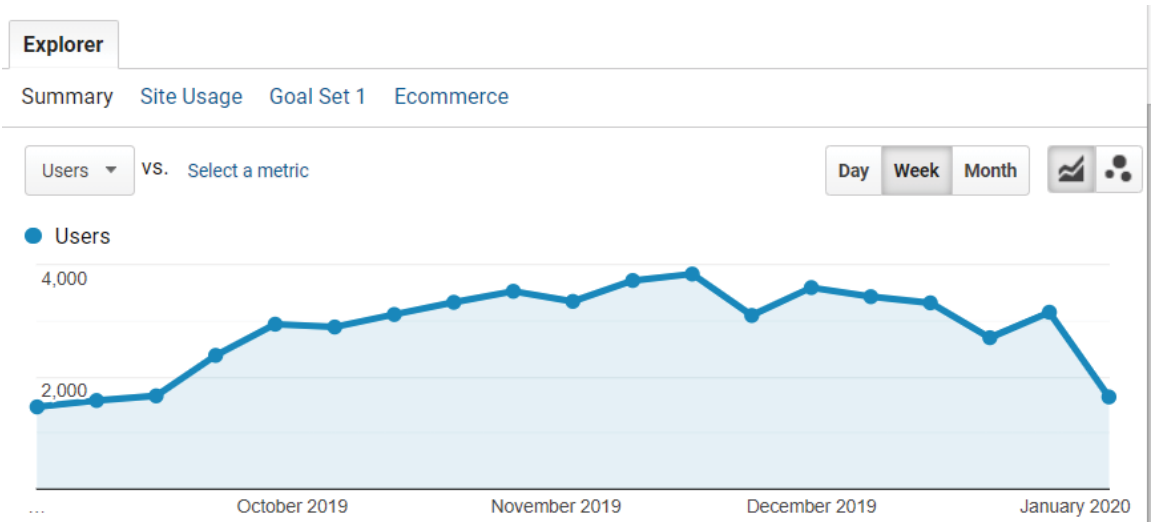
Improved Rankings In & Traffic From Google

There is a lot of traffic out there. The keyword "how do colorblind glasses work" is searched more than 1,300 times per month in the United States and 150 times per month in Canada. Add in spin-offs (such as "do colorblind glasses work"), and those numbers climb significantly.



Go ahead and do a search for that keyword. Notice how two eye care practices appear? That could be you. After all, who better to answer that question than an optometrist?

What happens when you write an article that gets cited, references, and shared? The graph tends to look something like this:



This Google Analytics screenshot showing weekly traffic was taken from an eye care practice in Maryland after the post went live in late September.

Note: This article was written in early January which is why January's numbers look so low by comparison.

Generate Leads... Lots of Leads

This is the most tangible (and important) benefit. Consider the graph from Google Analytics shown above: even if only 0.05% of the increased traffic resulted in a new patient or lead, for the above business, that means they are generating as many as 20 new leads per month, all from a single blog post.

How would that impact your business?

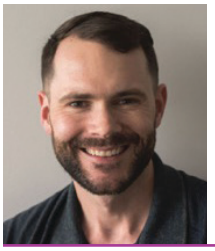
INVEST IN YOUR BLOG & REAP THE BENEFITS

Blogging is a simple method that has a shallow learning curve. Invest in great content that is deep and rich in detail and then share it on your social channels. With consistency, you'll see your rankings climb and new customers walk through the front door. **That is why you should be blogging.** ●

Remarque: ces articles ne sont pas examinés.

P GESTION DE CABINET

Pourquoi les optométristes devraient-ils commencer un blogue?



Cameron Martel
Cameron Martel est un spécialiste du marketing numérique chevronné qui gère des campagnes de SEO et de contenu depuis 2005. Il travaille actuellement avec des dizaines de cabinets de soins oculovisuels dans le cadre de son travail avec Marketing4ECPs. Il est daltonien, mais ne le lui rappelez pas, sinon il verra rouge! (ou du moins, c'est ce qu'il croit). Pour joindre Cameron : www.marketing4ecps.com ou cameron@4ecps.com.

Nous encourageons fortement les cabinets d'optométrie à créer un blogue sur les pratiques au sein de l'industrie. Il s'agit de l'un des moyens les plus efficaces de générer du trafic supplémentaire, d'accroître la visibilité de votre marque et d'attirer de nouveaux patients. C'est pourquoi nous en avons fait l'un des piliers de notre stratégie de SEO (*search engine optimization*, ou optimisation pour les moteurs de recherche en français).

De nos jours, les blogues sont moins en avant-plan, et ceux qui en rédigent s'y prennent souvent de la mauvaise manière. Bien des cabinets concentrent davantage leurs efforts sur les médias sociaux et ajoutent rarement, pour ne pas dire jamais, du nouveau contenu sur leur blogue. Voyons ensemble pourquoi il s'agit d'une occasion ratée pour eux, et d'une occasion en or pour vous.

POURQUOI LE BLOGUE EST-IL L'UN DES OUTILS DE RÉFÉRENCEMENT NATUREL LES PLUS PUISSANTS?

Google raffole du contenu de qualité. Google adore lorsque ses utilisateurs se perdent dans les dédales des résultats de recherche, enchaînant les blogues l'un après l'autre et absorbant ainsi de grandes quantités d'informations.

Pourquoi?

Parce que cela signifie que ses utilisateurs trouvent utiles les résultats que Google leur propose, et que si c'est réellement le cas, ces derniers reviendront encore et encore. Compte tenu du fait que Google détient plus de 90 % des parts du marché des moteurs de recherche, nous avons des raisons de croire que sa logique est implacable.

Il s'agit également de l'un des moyens les moins coûteux de cibler des clients potentiels. Par ailleurs, si vous créez le blogue vous-même, il vous en coûtera uniquement un peu de votre temps. Lorsqu'il est géré correctement, un blogue peut réellement transformer les activités d'une entreprise. Sans l'ombre d'un doute!

LES POSSIBILITÉS OFFERTES PAR LE BLOGUE

Les gens qui sous-estiment les blogues oublient deux points importants : en premier lieu, les utilisateurs *veulent* accéder à cette information et cherchent activement à l'obtenir; deuxièmement, Google *souhaite* que ces renseignements proviennent d'*experts en la matière* comme vous. En fait, Google a apporté un changement important à son algorithme en août 2018 afin de privilégier le contenu provenant de professionnels de la santé dans les résultats de recherche associés à des mots-clés relatifs à la médecine. Autrement dit, les optométristes disposent d'un avantage important en ce qui a trait aux mots-clés concernant la santé oculaire.

Les blogues comportent quatre principaux avantages d'un point de vue opérationnel :

Ils vous permettent de renseigner et de sensibiliser vos futurs patients et clients.
Combien de fois avez-vous dû justifier le prix d'un examen de la vue ou d'une monture? La réalité est que la plupart des gens sont tout simplement inconscients des choses qu'ils ignorent. Par l'entremise de votre blogue, vous pouvez informer vos lecteurs au sujet de la santé oculovisuelle, de la correction de la vue et des articles de lunetterie et tout ce qui s'en rapporte.

Un patient renseigné est plus susceptible de reconnaître la valeur de ce que vous lui proposez et beaucoup moins enclin à faire des achats en se fondant uniquement sur le prix.

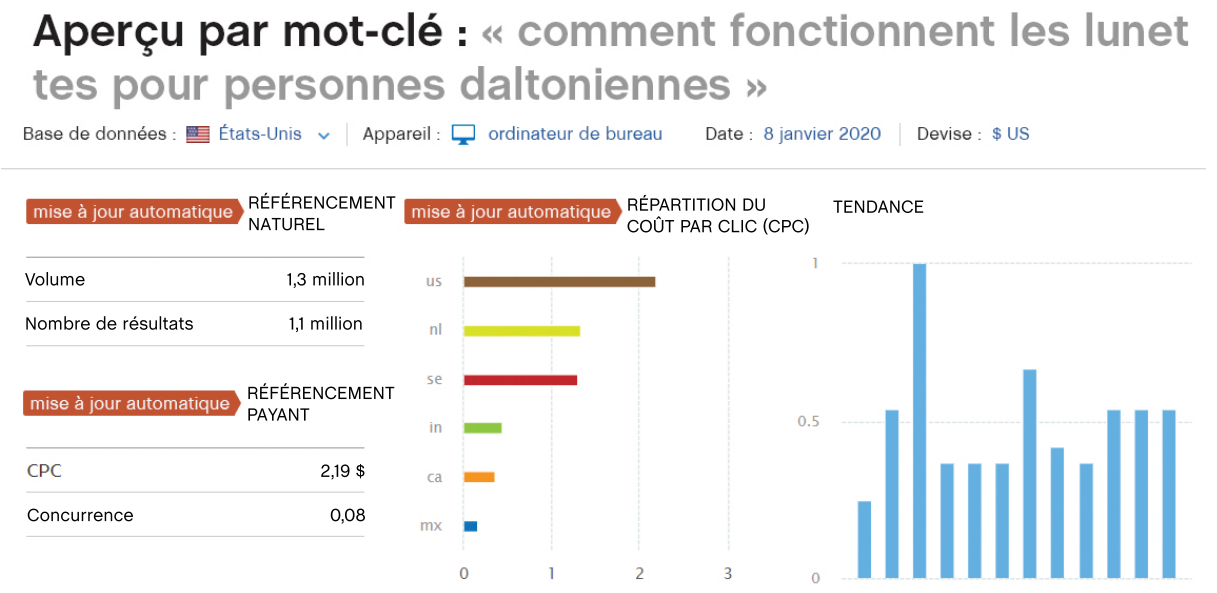
Ils vous permettent de démontrer votre expertise et votre leadership.

À l'ère des fausses nouvelles et de la désinformation, les gens cherchent à obtenir des renseignements auprès d'experts auxquels ils peuvent véritablement faire confiance. Si vous parvenez à les informer sur un sujet qui leur tient à cœur, vous gagnerez leur confiance.

Le contenu que vous créez pour votre blogue peut facilement être partagé sur vos réseaux sociaux, y compris LinkedIn. En répondant aux questions de vos lecteurs, vous gagnerez leur confiance et leur appui. Voilà quelque chose de puissant que vous ne pouvez accomplir par l'entremise de la publicité traditionnelle.

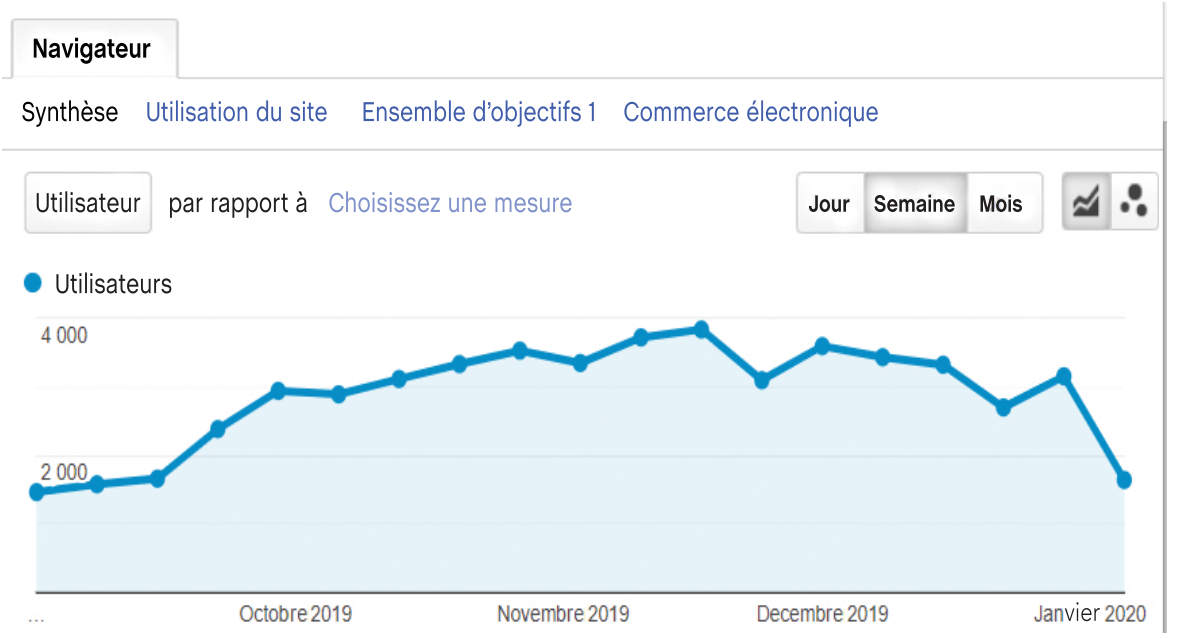
Ils vous permettent d'améliorer votre référencement dans Google et le trafic qu'il génère.

Il y a énormément de trafic sur le Web. La phrase « comment fonctionnent les lunettes pour personnes daltoniennes » fait l'objet d'une recherche plus de 1 300 fois par mois aux États-Unis et 150 fois par mois au Canada. Ajoutez à cela les différentes variantes (telles que « les lunettes pour personnes daltoniennes sont-elles efficaces ») et les chiffres augmentent de manière significative.



Allez-y, faites une recherche avec ce mot-clé. Avez-vous remarqué que deux cabinets d'optométrie figurent parmi les résultats? Ce pourrait être le vôtre. Après tout, y a-t-il une personne mieux placée qu'un optométriste pour répondre à cette question?

Que se passe-t-il lorsque vous écrivez un article et que celui-ci est cité, référencé et partagé? Le graphique tend à ressembler à ce qui suit :



Cette capture d'écran, tirée de Google Analytics, montre le trafic hebdomadaire sur le blogue d'un cabinet d'optométrie du Maryland après la publication d'un article à la fin du mois de septembre.

Note : Cet article a été rédigé au début du mois de janvier. C'est pourquoi les chiffres de janvier semblent si bas en comparaison avec ceux des autres mois.

Ils vous permettent de générer des clients potentiels… beaucoup de clients.

Il s'agit de l'avantage le plus concret (et le plus important). Jetez un coup d'œil au graphique ci-dessus tiré de Google Analytics : même si seulement 0,05 % du nouveau trafic permettait d'aller chercher un nouveau client ou de cibler un client potentiel pour l'entreprise présentée plus haut, celle-ci aurait généré jusqu'à 20 nouvelles pistes de clients potentiels par mois à partir d'un seul article de blogue.

Quelle en serait l'incidence pour votre entreprise?

CONSACREZ DU TEMPS À VOTRE BLOGUE ET RÉCOLTEZ LES FRUITS DE VOTRE LABEUR

La tenue d'un blogue est une méthode simple qui nécessite une courte période d'apprentissage. Consacrez du temps à la création de contenu de qualité riche en détail et partagez-le ensuite sur vos réseaux sociaux. Avec de la persévérance, vous obtiendrez un meilleur référencement et verrez de nouveaux clients franchir votre porte. **Voilà pourquoi vous devriez commencer un blogue.** ●

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