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VOL 74 NO 4 | 2012

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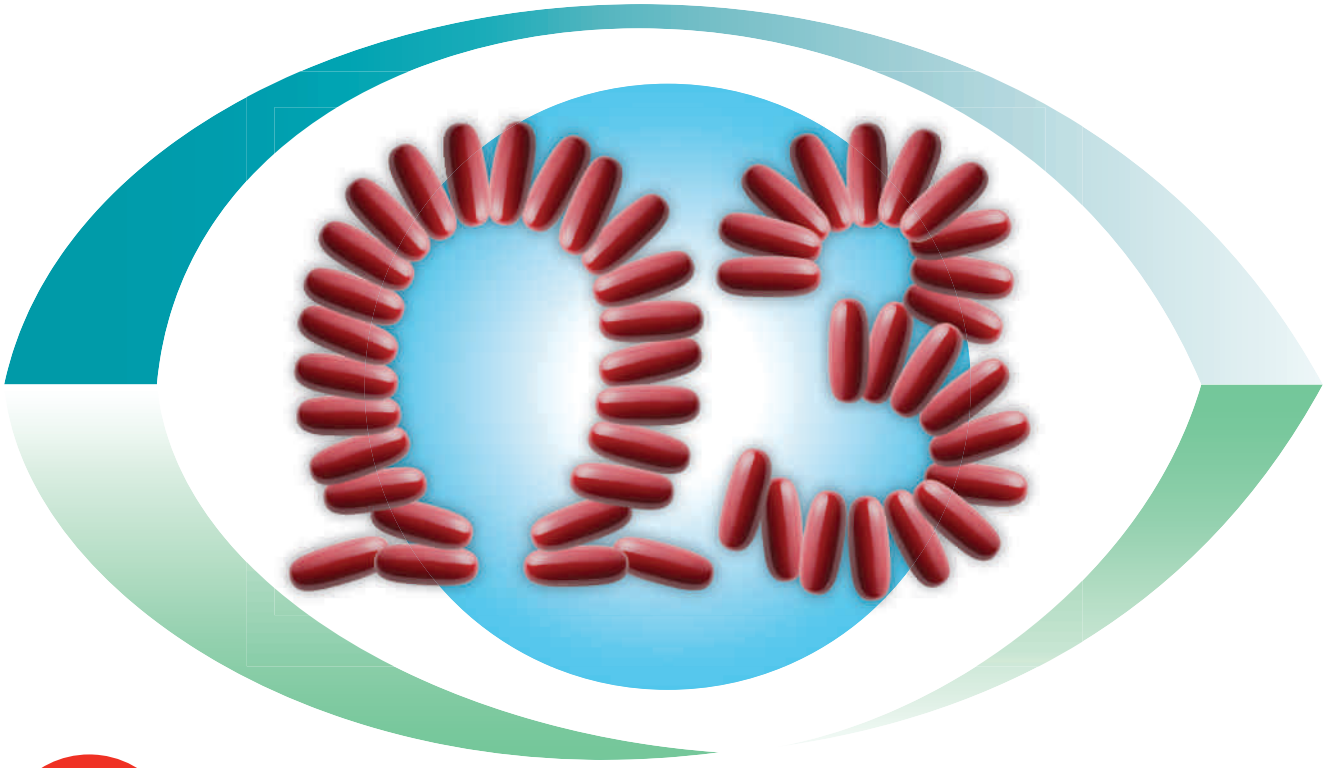
New Advancements in Nutrition for **Eye Health.**



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BY / PAR DR. LIL LINTON, OD

December 2012

It has taken over a year and a half for the latest iteration of our attempts to have non-corrective (cosmetic) contact lenses classified as medical devices to make its way through parliament to the point of approval. When all is said and done you could have completed an MBA program in the time that it will have taken for the bill to come into force. By all accounts this has been a fairly rapid process for such a simple bill. Given a more complex or controversial bill, the length of time to progress through each legislative step will increase to the point where one could likely obtain a baccalaureate degree before its progress is complete. Suffice it to say that changing legislation at any level is a time consuming and laborious process.

This year the CAO has worked with varying federal government agencies including Health Canada, Canada Revenue Agency, Human Resources and Skills Development Canada, Industry Canada, Corporations Canada, Citizenship and Immigration Canada, Canadian Institute for Health Information and the Public Health Agency of Canada. We have discussed issues around non-corrective contact lenses, non-insured health benefits, interim health benefits, grants and other issues important to eye health and the optometric profession. We have developed relationships with MPs, senators, bureaucrats and staff assistants. It has been a very active year in government relations for the CAO.

Government advocacy and relations is also a pressing need at the provincial level. Regardless of the different regulations that exist provincially, one commonality exists in that every provincial optometry association in Canada has pursued advocacy on one issue or another in their province — government relations and the efforts that go into trying to achieve change are substantial.

We know that changing legislation after it has been instituted is an incredibly difficult task. Every association's efforts are best placed in preventing change that is not in the public interest before it takes place. Being proactive requires effort that affects change when the policy makers are most receptive. Those efforts should focus on building strategic relationships, advocating policy positions, and educating policy influencers and decision makers.

There are many issues taking place provincially that should be on the national radar. Governments take their cues for policy positions from the public, interest groups, industry, and other governments. Policies being discussed in Saskatchewan and Manitoba can easily catch the attention of the Alberta or Ontario governments. Optometry must be constantly aware of policies and issues being discussed in other provinces, the United States, and other countries. In the information age, the world is too small for distance to be a barrier for domestic or foreign policies to set root.

Fortunately, there is a vehicle that exists within CAO to be able to discuss opportunities, issues and needs at the policy level and strategize plans to advance them. That vehicle is the CAO Government Relations Committee. It exists to bring provincial perspectives together so that we can discuss issues and opportunities, unite and proactively move forward together. It is the forum where we bring our collective knowledge, experience and GR resources together to affect change.

A primary role of the government relations committee is to determine initiatives that educate and influence government decision makers. CAO and the provincial associations need to take advantage of what the government relations committee offers; to monitor information, educate decision makers, advocate policy positions and work for change. This requires dynamic initiatives to bring eye health and optometry to the forefront through co-operative and strategic efforts within our profession.

We know how long policy change or creation can take. If we want changes within the next five years, efforts need to begin now.

Il a fallu plus d'un an et demi pour que la dernière édition des efforts que nous faisions pour que les lentilles cornéennes non correctives (cosmétiques) soient reconnues comme dispositifs médicaux en arrivent au stade de l'approbation au Parlement. Tout compte fait, il aurait été possible de terminer un programme de MBA pendant le temps qu'il aura fallu au projet de loi pour devenir loi. L'exercice a malgré tout été relativement rapide pour un projet de loi aussi simple. Le temps qu'il faut à une mesure plus complexe ou controversée pour franchir chaque étape législative augmente au point où il serait possible d'obtenir un baccalauréat avant que l'étude en soit terminée. Il suffit de dire que le changement d'une mesure législative à n'importe quel niveau est chronophage et laborieux.

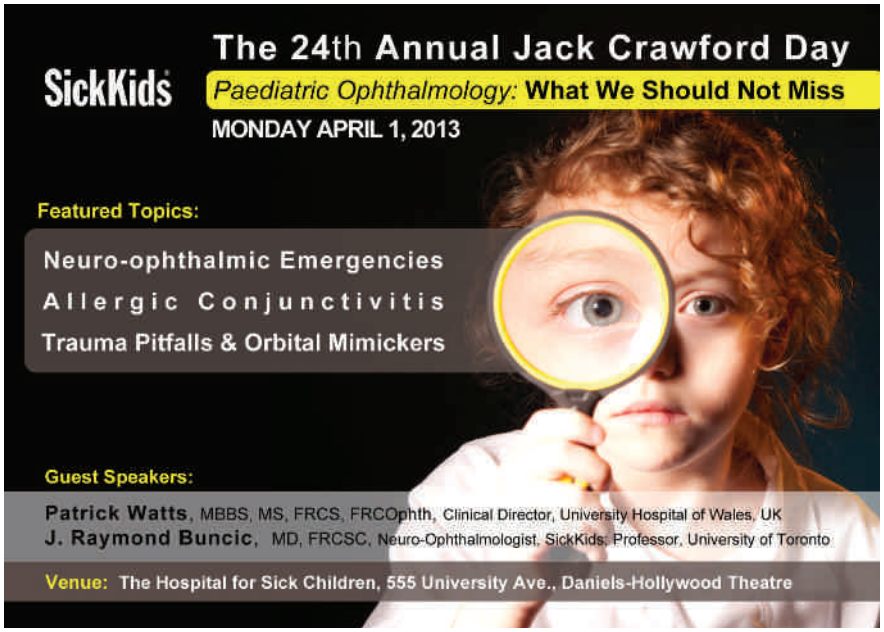
Cette année, l'ACO a collaboré avec divers organismes du gouvernement fédéral, y compris Santé Canada, l'Agence du

revenu du Canada, Ressources humaines et Développement des compétences Canada, Industrie Canada, Corporations Canada, Citoyenneté et Immigration Canada, l'Institut canadien d'information sur la santé et l'Agence de la santé publique du Canada. Nous avons discuté des lentilles cornéennes non correctives, des services de santé non assurés, des services de santé intérimaires, des subventions et d'autres questions importantes pour la santé oculovisuelle et la profession optométrique. Nous avons établi des liens avec des députés, des sénateurs, des fonctionnaires et des adjoints. L'année a été très active pour l'ACO sur le plan des relations gouvernementales.

La représentation et les relations gouvernementales constituent aussi un besoin pressant à l'échelon provincial. Sans égard aux différents règlements en vigueur dans les provinces, il existe un aspect commun, soit que les associations d'optométrie de chaque province du Canada sont intervenues dans un grand dossier ou un autre dans leur province – les relations gouvernementales et les efforts déployés pour essayer d'instaurer le changement sont importants.

Nous savons qu'il est incroyablement difficile de modifier une loi en vigueur. Il est préférable que les efforts de chaque association visent à prévenir au préalable un changement qui n'est pas dans l'intérêt public. Pour être proactif, il faut faire des efforts qui ont une incidence sur les changements lorsque les responsables des politiques sont les plus réceptifs. Ces efforts doivent viser avant tout à établir des liens stratégiques, défendre des positions stratégiques et informer les stratèges et les décideurs.

Il y a beaucoup de questions à l'échelon provincial qui devraient être visibles sur la scène nationale. Lorsqu'il s'agit d'arrêter des positions stratégiques, les gouvernements sont à l'écoute du public, des groupes spécialisés, de l'industrie et d'autres gouvernements. Des politiques qui font l'objet de discussions en Saskatchewan et au Manitoba peuvent facilement attirer l'attention des gouvernements de l'Alberta ou de l'Ontario. L'optométrie doit être constamment à l'affût des politiques et des enjeux abordés dans d'autres provinces, aux États-Unis et ailleurs. À l'ère de l'information, le monde est trop petit pour que les distances empêchent les politiques nationales ou étrangères de s'implanter.



Il existe heureusement à l'ACO un organe qui permet de discuter de possibilités, d'enjeux et de besoins à l'échelon stratégique et d'établir des plans stratégiques pour faire avancer ces dossiers. Cet organe est le Comité des relations avec les gouvernements de l'ACO qui doit unir les points de vue des provinces afin que nous puissions discuter d'enjeux et de possibilités, faire front commun et aller de l'avant ensemble de façon proactive. C'est la tribune où nous réunissons nos connaissances, notre expérience et nos ressources collectives pour instaurer le changement.

Le comité des relations avec les gouvernements, a pour rôle premier de déterminer les initiatives qui informent et influencent les décideurs des gouvernements. L'ACO et les associations provinciales doivent profiter de ce que le comité des relations avec les gouvernements peut leur offrir, surveiller l'information, informer les décideurs, préconiser des positions stratégiques et chercher à instaurer le changement. Il faut à cette fin des initiatives dynamiques pour placer la santé oculovisuelle et l'optométrie à l'avant-scène par des efforts stratégiques et basés sur la coopération à l'intérieur même de la profession.

Nous savons combien de temps il faut pour créer ou modifier une politique. Si nous voulons instaurer des changements au cours des cinq prochaines années, il faut nous mettre à l'œuvre maintenant.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Another year will soon be behind us, 2012 will soon be just a memory – time really does fly. My husband Dwight and I will be joining our family in Calgary for the holidays and flying to California on boxing day for some rest and recreation. I encourage all of you to take time to relax, enjoy your family, remember to count your blessings and share with those less fortunate. The Linton family wishes our optometric family across the country health, happiness and prosperity in 2013. I look forward to working with all of you in 2013 as we continue to move the profession of optometry forward.

Une autre année sera bientôt chose du passé, car 2012 ne sera bientôt plus qu'un souvenir – le temps file vraiment. Mon mari Dwight et moi-même allons nous joindre à notre famille à Calgary pour les vacances et nous nous envolerons pour la Californie le lendemain de Noël afin d'y trouver un peu de repos et de détente. Je vous encourage tous à prendre le temps de vous détendre, à profiter de votre famille, à ne pas oublier de compter vos bénédictions et de partager avec les moins fortunés. La famille Linton souhaite à notre famille optométrique d'un bout à l'autre du Canada santé, bonheur et prospérité en 2013. J'ai hâte de collaborer avec vous tous en 2013 pendant que nous continuons de faire avancer la profession.

– Dr. Lil Linton, CAO President / La Dre Lil Linton, présidente de l'ACO

Blue Cross Announcement

As of September 17, 2012, Medavie Blue Cross will begin introducing a new two-sided plastic identification card, similar to those used for banking. The new card design will be provided to new Medavie Blue Cross members as well as to any existing members when and if they require a change in their current ID card information such as name or dependent status.

Medavie Blue Cross is taking a staggered approach to introducing this new ID card and will not be replacing all cards currently in use. Many Medavie Blue Cross members will continue to use a four-sided blue laminated card. In Quebec, a two-sided yellow plastic card also exists. Please note that federal program members (RCMP, Veterans Affairs, Canadian Forces and Citizenship and Immigration Canada) do not carry Medavie Blue Cross cards.

Providers may call the Customer Service Centre toll-free if they have further questions: 1-800-667-4511 (in Atlantic), 1-800-355-9133 (in Ontario).1-888-588-1212 (in Quebec). Providers in other provinces please contact your local Blue Cross.

Annonce sur la Croix-Bleue

Le 17 septembre 2012, Croix Bleue Medavie a lancé une nouvelle carte d'identité en plastique à deux côtés, semblable aux cartes bancaires. La nouvelle carte sera fournie aux nouveaux membres de Croix Bleue Medavie, ainsi qu'à tout membre actuel qui doit modifier l'information contenue sur sa carte d'identité actuelle, comme son nom ou son statut de personne à charge. Croix Bleue Medavie met cette nouvelle carte d'identité en service de façon décalée et ne remplacera pas toutes les cartes en vigueur. Beaucoup de membres de Croix Bleue Medavie continueront d'utiliser une carte laminée bleue à quatre côtés. Au Québec, une carte en plastique jaune à deux côtés existe aussi. Il faut noter que les membres du programme fédéral (GRC, Anciens Combattants, Forces canadiennes et Citoyenneté et Immigration Canada) n'ont pas de carte Croix Bleue Medavie.

S'ils ont d'autres questions, les fournisseurs peuvent appeler gratuitement le Centre des services à la clientèle : 1-800-667-4511 (Atlantique), 1-800-355-9133 (Ontario), 1-888-588-1212 (Québec). Les fournisseurs des autres provinces sont priés de communiquer avec leur Croix Bleue locale.

Eyefoods Book Offer

Thank your faithful patients, referring doctors, or staff and show them your appreciation with a gift of good health this holiday season. The Eyefoods book makes a fitting present and an acknowledgment of their patronage and support throughout the year. Show your gratitude and take advantage of our special holiday offer: or kick start January's "get healthy" resolutions and sell Eyefoods books in your practice

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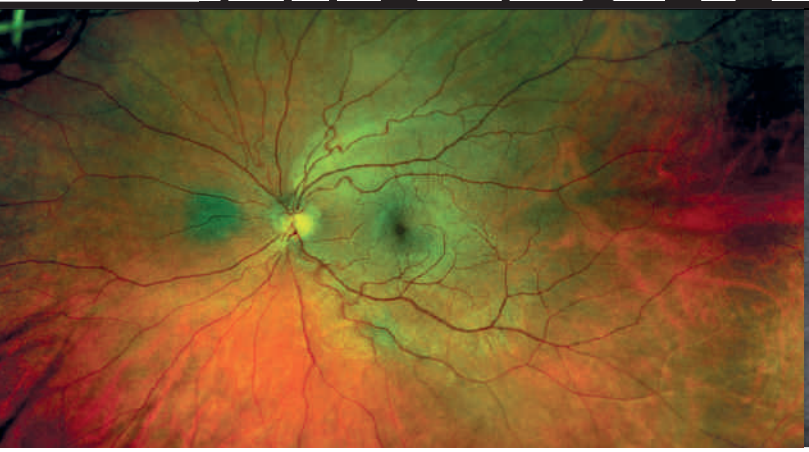

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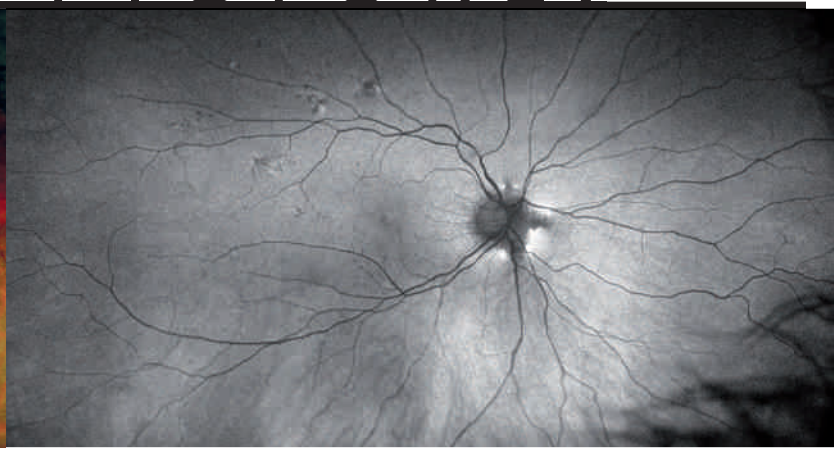

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Remerciez vos patients fidèles, les médecins qui les réfèrent ou les membres du personnel en leur témoignant votre appréciation par un cadeau de bonne santé au cours de la saison des Fêtes. L'ouvrage Eyefoods constitue un cadeau bien adapté et reconnaît leur clientèle ou leur soutien tout au long de l'année. Montrez-leur votre gratitude et profitez d'une offre spéciale des Fêtes, ou lancez rapidement les résolutions « de bonne santé » de janvier et vendez les ouvrages Eyefoods dans votre cabinet au montant de 24,95 $. Cliquez sur le lien de l'ouvrage au magasin en ligne d'Eyefoods. Achetez un carton de 36 ouvrages au prix de 13 $ l'unité (468 $ plus taxes et frais de port). Ce montant représente au total 1 195 $ de moins que le prix de détail!
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New Canada Not-For-Profit Corporations Act

On October 17, 2011 the Federal Government enacted the new Not-For-Profit Corporations Act and all federally incorporated not-for-profit organizations have 3 years to transition to the new act. This comprehensive act provides a foundation for the internal management of not-for-profit corporations. The transition process involves a review of the articles of incorporation and by-laws and to adopt changes to comply with the new act. The CAO studied the requirements to transition with the intent of presenting the necessary changes for member adoption at the next business meeting of members in Edmonton in July 2013. The timeline was recognized as very tight. The CAO was originally incorporated under a Special Act of Parliament. Special Act organizations are not required to transition by the October 17, 2014 deadline. Therefore, the CAO will take the time it has available to fully investigate a transition to the new act. CAO Council still expects to recommend several by-law changes at the general business meeting in Edmonton. Further information will be brought forward in the near future.

Nouvelle loi canadienne sur les organisations à but non lucratif

Le 17 octobre 2011, le gouvernement fédéral a adopté la nouvelle Loi canadienne sur les organisations à but non lucratif. Tous les organismes sans but lucratif qui ont une charte fédérale ont trois ans pour s'y conformer. Cette loi détaillée jette les bases de la gestion interne des personnes morales sans but lucratif. Le processus de transition comporte une revue des statuts constitutifs et des règlements et l'adoption de modifications pour devenir conforme à la nouvelle loi. L'ACO a étudié les exigences relatives à la transition afin de soumettre les changements qui s'imposent aux membres pour qu'ils les adoptent au cours de leur prochaine séance de travail qui aura lieu à Edmonton en juillet 2013. Le calendrier est très serré. L'ACO a été constituée à l'origine en vertu d'une loi spéciale du Parlement. Les entités constituées en vertu d'une loi spéciale ne sont pas tenues d'effectuer la transition au plus tard à la date limite, fixée au 17 octobre 2014. L'ACO prendra donc le temps mis à sa disposition pour étudier à fond le virage vers la nouvelle loi. Le Conseil de l'ACO s'attend quand même à recommander plusieurs modifications des règlements au cours de la séance de travail générale à Edmonton. D'autres renseignements vous parviendront sous peu.

Director Announcement

Waterloo's School of Optometry & Vision Science is pleased to announce that Professor Paul Murphy, BSc, FCOptom, PhD, FAAO, FBCLA, FEAOO will be the school's next director. Dr. Murphy is an optometrist (Cardiff), with a PhD (Glasgow) in ocular surface sensation and a postgraduate certificate in tertiary level teaching methods. He is currently working toward acquiring his MBA from the University of Glamorgan. He was previously a lecturer in the Department of Vision Sciences (Glasgow) and is currently Reader and Director of Teaching at the School of Optometry and Vision Sciences at Cardiff University. The attached link to the school's web-site provides a brief description of Dr. Murphy's career to date – uwaterloo.ca/optometry-vision-science/news/director-announcement. The school's interim administration will continue its work through to Professor Murphy's arrival.

Nomination d'un directeur annoncée

L'École d'optométrie et des sciences de la vision de Waterloo est heureuse d'annoncer que le Pr Paul Murphy, BS, FCOptom, PhD, FAAO, FBCLA, FEAOO, sera le prochain directeur de l'École. Le Dr Murphy est optométriste (Cardiff) et titulaire d'un doctorat (Glasgow) en sensation des surfaces oculaires et d'un certificat postdoctoral en méthodes d'enseignement au niveau tertiaire. Il prépare actuellement son MBA de l'Université de Glamorgan. Auparavant chargé de cours au Département des sciences de la vision (Glasgow), il est actuellement maître de conférence et directeur de l'enseignement à l'École d'optométrie et des sciences de la vision de l'Université de Cardiff. Le lien ci-joint vers le site Web de l'école donne accès à une brève description de la carrière du Dr Murphy jusqu'à maintenant - uwaterloo.ca/optometry-vision-science/news/director-announcement. L'administration intérimaire de l'école poursuivra son travail jusqu'à l'arrivée du Pr Murphy.

CCOHS Podcast

Dr. Cheryl Zimmer, Interim Director, Third Party Plans, CAO participated in an interview with the Canadian Centre for Occupational Health and Safety for a podcast about Computer Vision Syndrome. CCOHS is promoting the podcast through its Health and Safety Report newsletter emailed to 32,000 subscribers and Liaison Report that goes to 11,000 subscribers. It is also being promoted through social media. You may listen to the podcast at this link: ccohs.libsyn.com/shedding-light-on-computer-vision-syndrome.

Balado du CCHST

La Dre Cheryl Zimmer, directrice intérimaires, Régimes de tiers, ACO, a participé à une entrevue avec le Centre canadien d'hygiène et de sécurité au travail pour un balado portant sur le syndrome de vision informatique. Le CCHST fait la promotion du balado dans son bulletin Rapport sur la santé et la sécurité envoyé par courriel à 32 000 abonnés et dans le bulletin Liaison, que reçoivent 11 000 abonnés. On en fait aussi la promotion dans les médias sociaux. Vous pouvez écouter le balado en suivant ce lien : ccohs.libsyn.com/shedding-light-on-computer-vision-syndrome.

New Optometry Forum

an email-based and commercially-independent forum restricted to Canadian optometrists and optometric educators (i.e. professors in a school of optometry). The COG delivers email posted to subscribers by other subscribers. The originators hope this simple resource will be helpful for colleagues across Canada to communicate anything relevant to optometric practice - clinical questions, eye-care news, practice management tips, etc. It is a forum for optometrists organized and operated by two Canadian optometrists, Peter Rozanec, OD & Glen Chiasson, OD. If you are interested in becoming a member, please let them know and spread the word. To join, send an email to: canadianoptometrygroup@gmail.com.

Nouveau Forum de l'optométrie

Le Canadian Optometry Group (COG) est un forum électronique et indépendant sur le plan commercial qui est réservé aux optométristes et aux éducateurs en optométrie du Canada (c.-à-d. aux professeurs des écoles d'optométrie). Le COG livre des messages électroniques aux abonnés affichés par d'autres abonnés. Les auteurs espèrent que cette ressource simple aidera des collègues de partout au Canada à diffuser tout ce qui est important pour la pratique de l'optométrie - questions cliniques, nouvelles sur les soins oculovisuels, conseils sur la gestion d'un cabinet, etc. Ce forum qui s'adresse aux optométristes est organisé et administré par deux optométristes canadiens, Peter Rozanec, OD, et Glen Chiasson, OD. Si vous souhaitez devenir membre, veuillez les en informer et faire passer le mot. Pour adhérer, envoyez un message électronique à : canadianoptometrygroup@gmail.com.

Website Updates

Recent website changes of note include the CAO history page, which now includes a copy of the wording of the Federal Act to Incorporate the Canadian Association of Optometrists, a gallery of CAO presidents, and dates/locations of CAO Congresses. Information about Occupational Vision Care programs has been moved from the Open your eyes micro site to opto.ca/ovp. This page includes links to provincial OVC/OVPs and tips for completing forms. For those members who participate in the Ontario OVP, there is also additional content found when clicking on the 'Ontario' link.

Incident Reporting

CAO reminds members to report patient incidents on the national incident reporting site. Add to your provincial total by reporting asymptomatic patients, invalid prescriptions, online ordering, sight tests, and cosmetic contact lenses.

Please support this effort! To report an incident, visit: **www.surveymonkey.com/s/ODincidentreport**

Déclaration des incidents

L'ACO rappelle aux membres de déclarer les incidents liés à des patients sur le site national de déclaration des incidents. Contribuez aux totaux de votre province en déclarant les patients asymptomatiques, les prescriptions non valides, les commandes en ligne, les tests de la vue et les lentilles cornéennes à but esthétique.

Veuillez appuyer cet effort! Pour signaler un incident, rendez-vous à : **http://www.surveymonkey.com/s/ODrapportincident**

Mises à jour de site Web

Des modifications récentes et dignes de mention du site Web comprennent la page sur l'histoire de l'ACO, qui inclut maintenant une copie du texte de la Loi fédérale constitutive de l'Association canadienne des optométristes, une galerie de portraits des présidents de l'ACO et les dates et lieux des congrès de l'ACO. L'information sur les programmes de soins professionnels de la vue a été transférée du microsite Ouvrez les yeux à opto.ca/rpv. Cette page comprend des liens vers les SPV/RPSV des provinces, ainsi que des conseils sur la façon de remplir les formulaires. Les membres qui participent au programme RPSV de l'Ontario y trouveront du contenu supplémentaire en cliquant sur le lien « Ontario ».

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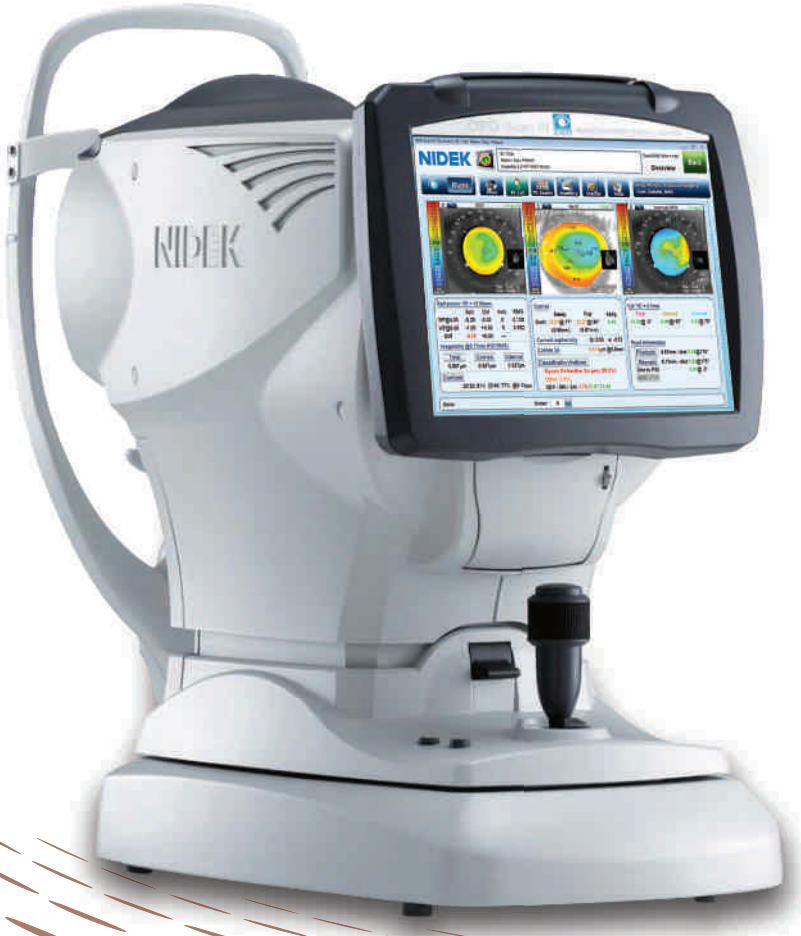
- Viewing software available for Exam Lane
- Pathology Discernment



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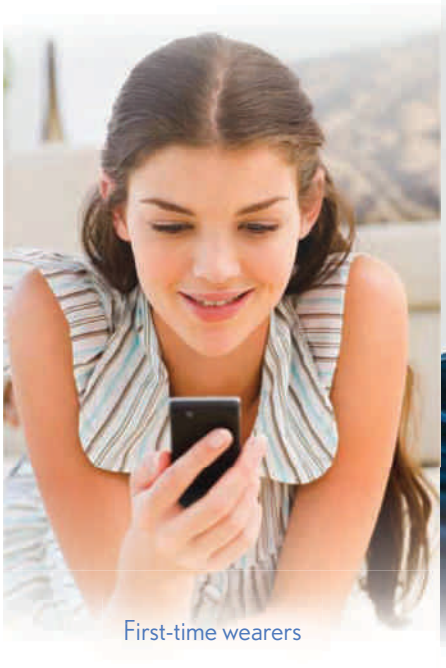
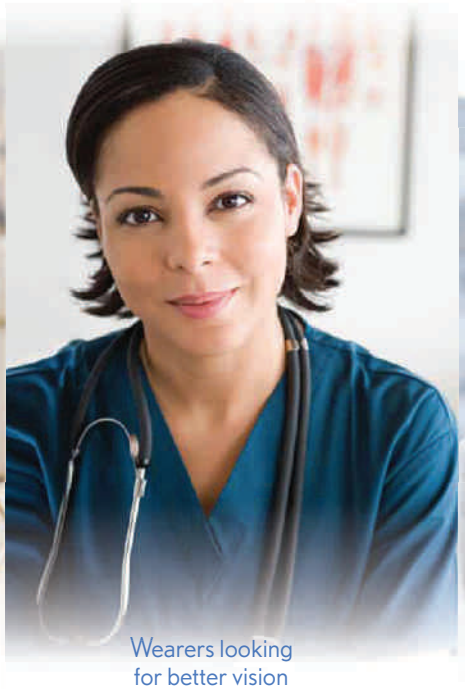
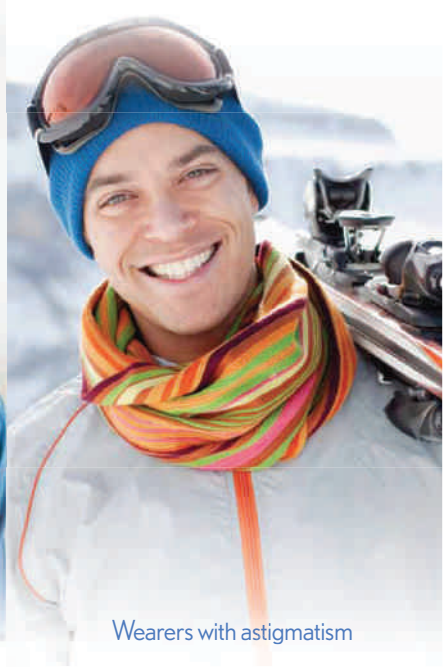


First-time wearers



Wearers looking for better vision



Wearers with astigmatism

Astigmatic contact lenses – A huge unmet need

You can be your patient's hero when they are in need of a new or updated vision prescription. But are all the options that might improve patient satisfaction – including, possibly, specialty contact lenses – receiving adequate consideration?

A recent publication evaluated a database of 11,624 spectacle prescriptions to calculate the prevalence of astigmatism of varying degrees and found that the prevalence of patients with astigmatism of 0.75 and 1.00 D or greater in at least one eye was 47.4% and 31.8%.[1] This represents a wealth of patients who may be coming to you for toric contact lenses. Yet, about four out of 10 astigmats who have never worn contacts have not tried them because of information from family, friends or something they read that people with astigmatism could not wear lenses. Even more amazing is that three out of 10 have not tried them due to advice from their doctor.[2]

Add this to the fact that globally, 43% of spectacle-wearing and 38% of contact-lens wearing patients with astigmatism reported less-than-complete satisfaction with the spectacles or contact lenses they wear most often, as judged by a score of 7 or less on a scale of 1 (very dissatisfied) to 10 (very satisfied).[3] Such findings point to unmet needs that could, in the case of astigmatic spectacle wearers, potentially be addressed by consideration of properly fitted toric contact lenses. The dissatisfaction among astigmatic contact lens wearers could similarly reflect a need for more appropriately selected and fitted lenses.

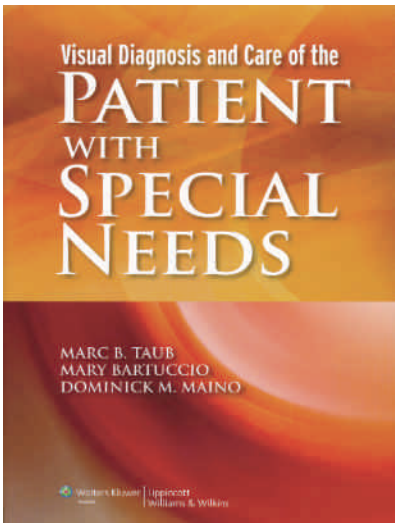
> You can be your patient's hero.

What are eye care professionals looking for in a toric lens to satisfy their astigmatic patients? In a study identifying the most important product attributes for eye care professionals when recommending soft toric contact lenses, the top 3 toric lens benefits related to vision, with the attribute of highest relative importance being "delivers crisp, sharp vision all day."[4] Likewise, when patients with astigmatism were asked about the top product attributes when selecting contact lenses, the top 5 toric lens attributes were visual benefits, with the benefit of highest relative importance being "delivers consistently clear vision at all times."[5]

The opportunity to fit and satisfy astigmatic patients is well within our reach; however there is a genuine need and opportunity for practitioners to proactively recommend and prescribe a more satisfying solution for their astigmatic patients. The good news is that eye care professionals and patients do agree that when it comes to selecting toric soft lenses, the importance of visual benefits rises to the top.

BAUSCH+LOMB

[1] Young G, Sulley A, Hunt C. Prevalence of astigmatism in relation to soft contact lens fitting. *Eye Contact Lens*. 2011 Jan;37(1):20-5. [2] Astigmatism: Incidence & Barriers. U.S. Market Research Report. *Decision Analyst*. December 2008. [3] Needs, Symptoms, Incidence, Global Eye Health Trends (NSIGHT) Study. Market Probe Europe. December 2009. [4] ECP Toric Needs Study: US. Millward Brown. December 2010. [5] Consumer Toric Needs Study: US. Millward Brown. December 2010.



Visual Diagnosis and Care of the Patient with Special Needs

BY CHERYL ZIMMER, OD, BSc

The miracles of modern medicine have allowed premature infants a 90% survival rate after only 27 weeks gestation, according to the March of Dimes.[1] Developmentally and intellectually delayed children now live to adulthood and seniors are living longer than ever imagined, often with multiple health issues. As optometrists, primary health care professionals, we have the responsibility to provide vision care to everyone, and we must be prepared for that endeavour. Visual Diagnosis and Care of the Patient with Special Needs written by Marc B. Taub, Mary Bartuccio and Dominick M. Maino, all doctors of optometry, is the essential resource for taking care of special populations.

The populations in this book include those that may be born with a disability or syndrome as a result of problems during gestation or genetic mutations, such as those with cerebral palsy, Down syndrome, fragile X syndrome as well as intellectual disabilities of unknown origins, and those special populations that have acquired illnesses such as brain injury from disease, accident or stroke, psychiatric disorders and neurodegenerative diseases. Each condition is discussed from a systemic standpoint and oculovisual anomalies and their clinical implications are addressed.

The management and treatment of those with autism spectrum disorders, attention deficit hyperactivity disorder and learning disabilities are also discussed at length. These conditions are more readily diagnosed than ever before and more prevalent in the class room and the primary care optometrist's examination room. The Canadian Association of Optometrists, Eye Health Month's slogan for 2012 was Look, See, Learn. This applies to all children, and this book equips the optometrist to better deal with those for whom vision and learning are not straightforward.

Some visual issues affecting these populations include refractive errors, strabismus, amblyopia and visual field defects, as well as oculomotor dysfunctions. Computers have provided patients with better access to visual rehabilitation, treatment and enhancement. These techniques and procedures are addressed in detail for both in-office assessment and home use.

One other area of interest discussed at length is the visual processing issues that special needs populations encounter that cannot be treated with traditional spectacle therapy. The book introduces additional procedures not commonly used during the conventional comprehensive assessment of the visual system, but necessary for the assessment of the non-verbal patient or those with multiple system delay.

One of the most intriguing chapters outlines the optometric management of functional vision disorders. Treatment includes the use of spectacles and prisms to relieve stress on the visual system and improve binocular performance. In conjunction, occlusion and vision therapy may also be implemented. Case examples are used to illustrate these management techniques. The neurological basis for vision therapy is also discussed in this chapter, stressing the integration of vision with other sensory inputs and how these interwoven systems play such a vital role in a person's localization and orientation in the world.

The authors stress a multi-disciplinary approach where the patient's needs are assessed and treated by a variety of specializations including, but not limited, to their physician, neurologist, ophthalmologist and optometrist as well as adjunct health care workers such as chiropractors, occupational therapists, social workers and dieticians. The objective is to reduce the total load of systemic and visual assault on the body experienced by special needs patients with many of these debilitating syndromes. Enhanced communication between the disciplines is in the best interest of the patient.

The Visual Diagnosis and Care of the Patient with Special Needs may be read from cover to cover, providing the reader with a wealth of incredible and pertinent information, or used as a reference manual in the primary care optometry office. The authors take into account that the readers of this publication are well versed in the procedures used during a routine eye examination, but they expand the reader's knowledge base and apply these diagnostic and treatment techniques to those with special needs. Each chapter has a multitude of informed contributors and is eloquently and concisely written. There are extensive references at the end of each chapter providing resources for those who may want more insight into certain topics. Congratulations to Marc B. Taub, Mary Bartuccio and Dominick M. Maino for providing our profession with such an outstanding and informative resource.

Endnotes

1 http://www.marchofdimes.com/baby/loss_neonataldeath.html

Target Seasonal Allergic Conjunctivitis with Alrex®

Treat the Signs and Symptoms

- ALREX® for temporary relief of the signs and symptoms of seasonal allergic conjunctivitis[1]
- Proven efficacy with an excellent safety profile[1]
- Available in 5 mL bottles

ALREX® (loteprednol etabonate) Ophthalmic Solution 0.2% is indicated for temporary short-term relief of the signs and symptoms of seasonal allergic conjunctivitis.

Alrex® is for ophthalmic, short-term use only (up to 14 days). If Alrex® is used for 10 days or longer, intraocular pressure should be monitored.

Alrex® is contraindicated in suspected or confirmed infections of the eye: viral diseases of the cornea and conjunctiva including epithelial *herpes simplex* keratitis (dendritic keratitis), vaccinia, and varicella; untreated ocular infection of the eye; mycobacterial infection of the eye and fungal diseases of ocular structures; hypersensitivity to this drug or any ingredient in the formulation or container, or to other corticosteroids.

Reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, secondary ocular infection from pathogens including *herpes simplex*, and perforation of the globe where there is thinning of the cornea or sclera.

In clinical studies, adverse events related to loteprednol etabonate were generally mild to moderate, non-serious and did not interrupt continuation in the studies. The most frequent ocular event reported as related to therapy was increased IOP: 6% (77/1209) in patients receiving loteprednol etabonate, as compared to 3% (25/806) in the placebo treated patients.

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Pr Alrex®
loteprednol etabonate
ophthalmic suspension 0.2%

PAAB

Bausch & Lomb Canada Inc., Vaughan, Ontario L4K 4B4

References: 1. ALREX Product Monograph, December 22, 2008

See prescribing summary on page 16

Pr Alrex®

(loteprednol etabonate ophthalmic suspension 0.2% w/v)

Prescribing Summary

Patient Selection Criteria

THERAPEUTIC CLASSIFICATION

Corticosteroid

INDICATIONS AND CLINICAL USE

Alrex® (loteprednol etabonate) Ophthalmic Suspension is indicated for temporary short-term relief of the signs and symptoms of seasonal allergic conjunctivitis

CONTRAINDICATIONS

Suspected or confirmed infection of the eye: viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella; untreated ocular infection of the eye; mycobacterial infection of the eye and fungal diseases of ocular structures; hypersensitivity to this drug or any ingredient in the formulation or container, or to other corticosteroids.

SPECIAL POPULATIONS

Use in Pediatrics (< 18 years of age):

Alrex® should not be used in pediatric patients.

Use in Geriatrics:

Alrex® should not be used in geriatric patients. The safety and efficacy of Alrex® have not been established in patients > 65 years of age.

Pregnant Women:

Alrex® should not be used in pregnant women, unless the benefit clearly outweighs the risks. Studies in pregnant women have not been conducted.

Nursing Women:

Alrex® should not be used in lactating women, unless the benefit clearly outweighs the risks.

Safety Information

WARNINGS AND PRECAUTIONS

General

For ophthalmic, short-term use only (up to 14 days).

The initial prescription and renewal of Alrex® should be made by a physician only after appropriate ophthalmologic examination is performed. If signs and symptoms fail to improve after two days, the patient should be re-evaluated. If Alrex® is used for 10 days or longer, intraocular pressure should be closely monitored.

Prolonged use of corticosteroids may result in cataract and/or glaucoma formation. Alrex® should not be used in the presence of glaucoma or elevated intraocular pressure, unless absolutely necessary and close ophthalmologic monitoring is undertaken. Extreme caution should be exercised, and duration of treatment should be kept as short as possible.

Alrex® should not be used in cases of existing (suspected or confirmed) ocular viral, fungal, or mycobacterial infections. Alrex® may suppress the host response and thus increase the hazard of secondary ocular infections. The use of Alrex® in patients with a history of herpes simplex requires great caution and close monitoring.

Alrex® contains benzalkonium chloride.

Alrex® has not been studied in pregnant or nursing women, but has been found to be teratogenic in animals. Alrex® should not be used in pregnant or nursing women unless the benefits clearly outweigh the risks.

Carcinogenesis and Mutagenesis

Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma tk assay, or in a chromosome aberration test in human lymphocytes, or *in vivo* in the single dose mouse micronucleus assay.

Ophthalmologic

Alrex® should be used as a brief temporary treatment. If Alrex® is used for 10 days or longer, intraocular pressure should be closely monitored. The initial prescription and renewal of Alrex® should be made by a physician only after appropriate ophthalmologic examination is performed, ie. slit lamp biomicroscopy or fluorescein staining if appropriate. If signs and symptoms fail to improve after two days, the patient should be re-evaluated.

Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision, and in posterior subcapsular cataract formation. Alrex® should not be used in the presence of glaucoma or elevated intraocular pressure, unless absolutely necessary and careful and close appropriate ophthalmologic monitoring (including intraocular pressure and lens clarity) is undertaken.

Corneal fungal infections are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration involving steroid use. Fungal cultures should be taken when appropriate.

Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In those diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids. In acute purulent conditions of the eye, steroids may mask infection or enhance existing infection.

Use of ocular steroids may prolong the course and may exacerbate the severity of many viral infections of the eye (including herpes simplex). Employment of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution.

Formulations with benzalkonium chloride should be used with caution in soft contact lens wearers.

ADVERSE REACTIONS

Overview

Reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, secondary ocular infection from pathogens including herpes simplex, and perforation of the globe where there is thinning of the cornea or sclera.

In nineteen clinical trials ranging from 1 to 42 days in length, 1,209 patients received various concentrations of loteprednol etabonate in topical ocular drops (0.005%, 0.05%, 0.1%, 0.2%, 0.5%). Adverse events related to loteprednol etabonate were generally mild to moderate, non-serious and did not interrupt continuation in the studies. The most frequent ocular event reported as related to therapy was increased IOP: 6% (77/1209) in patients receiving loteprednol etabonate, as compared to 3% (25/806) in the placebo treated patients.

With the exception of elevations in IOP, the incidence of events in the LE group was similar to, or less than that of the placebo control groups. Itching was reported as related to therapy in 3% of the loteprednol treated eyes, injection, epiphora, burning/stinging other than at instillation, foreign body sensation, and burning/stinging at instillation were each reported for 2% of eyes. The most frequent non-ocular event reported as related to therapy was headache, reported for 1.2% of the loteprednol treated subjects and 0.6% of the placebo treated subjects.

To report an adverse event, contact your Regional Adverse Reaction Monitoring Office at 1-866-234-2345 or Bausch & Lomb at 1-888-459-5000

Administration

One drop instilled into the affected eye(s) four times daily for up to 14 days. If scheduled dose is missed, patient should be advised to wait until the next dose and then continue as before.

SHAKE VIGOROUSLY BEFORE USING. Alrex® should be stored upright between 15°–25°C for up to 28 days after first opening.

The preservative in Alrex®, benzalkonium chloride, may be absorbed by soft contact lenses, and can discolour soft contact lenses. Therefore, Alrex® should not be used while the patient is wearing soft contact lenses. Patients who wear soft contact lenses and whose eyes are not red should wait ten to fifteen minutes after instilling Alrex® before they insert their contact lenses.

Patients should be advised not to wear a contact lens if their eye is red. Alrex® should not be used to treat contact lens related irritation.

SUPPLEMENTAL PRODUCT INFORMATION

WARNINGS AND PRECAUTIONS

Sexual Function/Reproduction

The effects of Alrex® on sexual function and reproduction have not been studied in humans. Treatment of male and female rats with up to 50 mg/kg/day and 25 mg/kg/day of loteprednol etabonate, respectively, (1000 and 500 times the Alrex® clinical dose) prior to and during mating, was clearly harmful to the rats, but did not impair their copulation

performance and fertility (i.e., ability of female rats to become pregnant). However, these doses were highly toxic and had significant toxic effects on the pregnancies, and the survival and development of the offspring. Maternal toxicity, possible occurrence of abnormalities and growth retardation started at 10 times the Alrex® clinical dose.

Neurologic
Disturbances and suppression of the Hypothalamic-Pituitary-Adrenal (HPA) axis can occur with systemic exposure to corticosteroids. However, given the very low systemic exposure to loteprednol etabonate when using Alrex® as directed, these possible effects are not likely.

Endocrine and Metabolism
Glucocorticoids, mostly when systemic exposure occurs, decrease the hypoglycemic activity of insulin and oral hypoglycemics, so that a change in dose of the antidiabetic drugs may be necessitated. In high doses, glucocorticoids also decrease the response to somatotropin. The usual doses of mineralocorticoids and large doses of some glucocorticoids cause hypokalemia and may exaggerate the hypokalemic effects of thiazides and high-ceiling diuretics. In combination with amphotericin-B, they also may cause hypokalemia. Glucocorticoids appear to enhance the ulcerogenic effects of non-steroidal anti-inflammatory drugs. They decrease the plasma levels of salicylates, and salicylism may occur on discontinuing steroids. Glucocorticoids may increase or decrease the effects of prothrombopenic anticoagulants. Estrogens, phenobarbital, phenytoin and rifampin increase the metabolic clearance of adrenal steroids and hence necessitate dose adjustments.
However, given the very low systemic exposure to loteprednol etabonate when using Alrex® as directed, these possible effects are not likely.

Immune
Cortisol and the synthetic analogs of cortisol have the capacity to prevent or suppress the development of the local heat, redness, swelling, and tenderness by which inflammation is recognized. At the microscopic level, they inhibit not only the early phenomena of the inflammatory process (edema, fibrin deposition, capillary dilation, migration of leukocytes into the inflamed area, and phagocytic activity) but also the later manifestations, such as capillary proliferation, fibroblast proliferation, deposition of collagen, and, still later, cicatrisation.

Clinical Trial Adverse Drug Reactions
Possibly or probably related adverse events from two Phase III studies are listed below:

	Alrex® 0.2% N = 133	Placebo N = 135
SPECIAL SENSES (EYE DISORDERS)		
Intraocular Pressure		
- elevation of 6 to 9mm Hg*	2% to 12%*	0% to 6%*
- elevation of ≥10mm Hg	1 (1%)	1 (1%)
Chemosis	6 (5%)	7 (5%)
Vision, Abnormal or Blurred	4 (3%)	5 (4%)
Burning/Stinging, on instillation	3 (2%)	6 (4%)
Itching Eye	3 (2%)	3 (2%)
Dry Eye	2 (2%)	4 (3%)
Burning/Stinging, not on instillation	2 (2%)	2 (1%)
Epiphora	1 (1%)	9 (7%)
Discharge	1 (1%)	3 (2%)
Foreign Body Sensation	1 (1%)	1 (1%)
Discomfort Eye	1 (1%)	0 (0%)
Injection	1 (1%)	0 (0%)
Eye Pain	1 (1%)	0 (0%)
Sticky Eye	0 (0%)	7 (5%)
Erythema Eyelids	0 (0%)	2 (1%)
Eye Disorder	0 (0%)	2 (1%)
BODY AS A WHOLE		
Face Edema (Head)	1 (1%)	0 (0%)
Allergic Reaction	1 (1%)	0 (0%)
MUSCULOSKELETAL SYSTEM		
Twitching	0 (0%)	1 (1%)

* for IOP increase of 6 to 9 mm Hg, please see below

One patient in the Alrex® group and one patient in the placebo group experienced increases in IOP of ≥10 mm Hg. Among these, one in each group had an IOP increase of ≥15 mm Hg, reaching IOP values over 30 mm Hg.
In both studies, there were more patients with IOP increases of 6 to 9 mm Hg in the Alrex® group than in the placebo group (see table below). In study A, among the patients with IOP increases of 6 to 9 mm Hg, four reached an IOP value of 22 to 23 mm Hg, and one patient reached 29 mm Hg and was discontinued (clinically significant increase in IOP). All these five patients were from the Alrex® groups.

Incidence of IOP increases of 6 to 9 mm Hg from baseline
(number of patients and percentages)

	Duration of treatment		
	Day 7	Day 14	Day 28
Alrex®			
Study-A	6 (9%)	6 (9%)	8 (12%)
Study-B	3 (5%)	1 (2%)	4 (6%)
Placebo			
Study-A	0 (0%)	4 (6%)	1 (2%)
Study-B	0 (%)	0 (%)	0 (%)

Due to the sample size for each arm of the two phase III studies in SAC, all events captured are greater than 1% of n.

SYMPTOMS AND TREATMENT OF OVERDOSAGE
For management of suspected accidental oral ingestion or drug overdose, consult your regional poison control centre.
No cases of overdose have been reported.
Full Product Monograph available for health professionals at: http://www.bausch.ca

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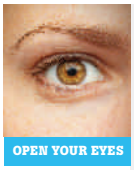
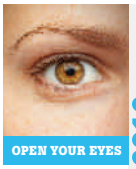
PAAB

THANK YOU | MERCI



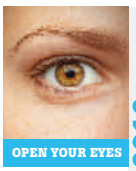
GOLD PARTNER 2012

Alcon
Johnson & Johnson Vision Care



SILVER PARTNER 2012

Centennial Optical
Essilor
Hoya Vision Care
Luxottica Group



BRONZE PARTNER 2012

Bausch & Lomb
CooperVision
Nikon Optical Canada
Scotiabank
Transitions
Carl Zeiss Canada

Introducing the iQ TrueForm Family
Introduisant la Famille iQ TrueForm

HOYA
PRACTICE MADE PERFECT

Hoya Vision Care's iQ Family of lenses, using TrueForm Lens Technology, offers the best of both worlds: the benefits of FreeForm design with an ideal solution to upgrade both progressive and single vision patients to an affordable custom option.

With TrueForm Lens Technology, HOYA applies FreeForm design principles to a semi-finished front surfaced lens. By applying additional aberration correction along each line of sight, supported by an aspheric/atoric back surface, the visual performance of the lens can be optimized over the entire lens for each individual prescription.

The iQ Summit ecp and cd, as well as the iQ Amplitude and iQ Amplitude Mini lenses take an existing premium progressive design, then uses FreeForm back surfacing and polishing techniques to optimize each prescription. Patients will experience noticeable improvements in peripheral vision at all distances, as well as easier adaptation and wider and clearer viewing ranges.

iQ Single Vision, the newest lens in the iQ TrueForm family, combines a spherical front surface single vision lens with HOYA FreeForm custom back surfacing for enhanced optical performance. iQ Single Vision lenses provide a better vision solution for patients, especially for those with higher prescriptions and/or astigmatism.

So the only question left is, what's your iQ?

.....

La famille de lentilles de Hoya Vision, utilisant la technologie TrueForm, offre le meilleur des deux mondes : les bénéfices d'un design FreeForm avec une solution idéale pour rehausser autant les patients porteurs de progressifs que de simple vision, et ce étant une option abordable.

Avec la technologie TrueForm, HOYA applique les principes de designs FreeForm à un semi-fini ayant un design de face avant. En appliquant une correction d'aberrations additionnelle à chaque ligne de vision, supportée par une surface interne asphérique/atorique, la performance visuelle de la lentille peut être optimisée sur toute la surface de la lentille et ce, pour chaque prescription individuelle.

La lentille iQ Summit ECP et CD, et la iQ Amplitude et iQ Amplitude Mini prennent un design progressif primé, puis utilisent les techniques de surfaçage et polissage FreeForm pour optimiser chaque prescription. Les patients expérimenteront des améliorations apparentes en vision périphérique à toutes les distances, ainsi qu'une adaptation plus facile avec une étendue plus large et claire de la vision.

La iQ Single Vision, la lentille la plus récente de la famille iQ TrueForm, combine une lentille sphérique simple vision sur la face avant à une face arrière sur mesure surfacée par la technologie HOYA FreeForm pour une performance optique rehaussée. Les lentilles iQ Single Vision offrent à vos patients une meilleure solution visuelle, spécifiquement pour les hautes prescriptions et/ou astigmatisme.

Alors, la question qui reste, quel est votre QI?

The COETF Annual Awards Program for 2012

The Canadian Optometric Education Fund (COETF) received a total 37 applications for awards in 2012. Of those 37 applications, 25 were granted at least partial funding for projects or research. In most cases, applicants are not given full funding as the total amount of funding requested greatly exceeds the money available for granting. Awards funding is based on the Trust Fund's interest earned over the previous year.

All award recipients are required to submit an interim report on their project and a final report upon completion. In an effort to recognize some of the projects and research being done by COETF award recipients the Awards Committee will publish project reports in the Canadian Journal of Optometry (CJO) so that our members across the country can learn more about where COETF funding goes, as well as highlighting exciting optometric research.

The COETF Annual Awards Program for 2012

SCHOOL OF OPTOMETRY AND VISION SCIENCE, UNIVERSITY OF WATERLOO

Stacey Chong (Master's Degree Program) *"Can retinal ganglion cells regenerate?"*

Brad Hall (PhD Program) *"Investigation of short term lactoferrin conformation on contact lenses using raman spectroscopy"*

Amith Hathibelagal (Master's Degree Program) *"A novel objective measure of infant visual acuity using gaze tracking"*

Charlene Hickey (Optometry student project) *"Tracking the development of binocular vision following spectacle correction in young children"*

Alex Hui (PhD Program) *"The effect of release solution replenishment on ciprofloxacin drug release from model silicone"*

Salsabeel Jadi (Master's Degree Program) *"The efficiency of multi-purpose solutions in removing protein from silicone hydrogel contact lens materials"*

Varadharajan Jayakumar (Master's Degree Program) *"Effect of source characteristics of an illuminated Placido disc on measurement of anterior surface aberrations"*

Holly Lorentz (Post-Doc Program) *"The efficacy of newer multipurpose cleaning solutions on lipid removal from silicone hydrogel contact lenses"*

Nicholas Lorentz (PhD Program) *"Investigation in vergence adaptation"*

Museum Of Visual Science *"Historical Archive/Museum Exhibit"*

Alan Ng (Master's Degree Program) *"Development of a novel fluorescent based lysozyme assay to study the conformational state of lysozyme deposited on contact lenses"*

William Ngo *(Master's Degree Program) "Imaging meibomian glands with OCT"*

Heinz Otchere *(Master's Degree Program) "Fitting semi-scleral lenses using corneal sagittal depth measurement and assessment of visual acuity"*

Chau-Minh Phan *(PhD Program) "Antifungal natamycin uptake and release in commercial contact lenses"*

Marc Schulze *"Determining the ocular shape in children and adults using optical coherence"*

Thanh Tran *(Master's Degree) "Characterization of retinal degeneration in Smoky Joe chickens"*

Chitman Uppal *(Master's Degree Program) "Relative choroidal and retinal vascular reactivity in diabetic retinopathy"*

Jalaiah Varikooty *(PhD Program) "Estimating in-vivo contact lens wettability through tear film hydrodynamics"*

Hendrik Walther *(Master's Degree Program) "Conformational state of meibum lipids in solution using raman spectroscopy"*

Gah-Jone Won *(Master's Degree Program) "The effects of non-muscle inhibitors on the biomechanics of the avian crystalline lens"*

QUICK FACTS

The COETF was created in 1976 by the members of the Canadian Association of Optometrists to assist programs in research, education and human resources development in the vision and eye care field in Canada. Through its annual program of Awards, the COETF has supported faculty development, research and/or specialized education programs carried out by graduate students and investigative projects conducted by undergraduates and faculty at Canada's schools of optometry, as well as projects undertaken by independent practitioners or members of the public.

Total Waterloo School of Optometry Applications	29	$ 164,650.98
Total Waterloo School of Optometry Awards	21	$ 38,000.00
Total Montréal École d'Optométrie Applications	6	$ 25,500.00
Total Montréal École d'Optométrie Awards	3	$ 8,500.00
Total Independent Practitioner Applications	2	$ 6,445.00
Total Independent Practitioner Awards	1	$ 1,000.00
Total Applications for 2012	37	$ 196,595.98
Total Awards for 2012	25	$ 47,500.00
Total Applications (since inception)		$6,417,867.76
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COEFT REPORTS

In an effort to highlight some of the projects and research by COETF award recipients, the COETF Trustees and Awards Committee have selected project reports to be published in the Canadian Journal of Optometry (CJO). Recognizing that many recipients intend to publish their work in cited journals, the reports are not considered to be clinical articles. COETF funded research, when completed and peer-reviewed may be published in the CJO and other journals. The COETF reports are intended to provide relevant information for the benefit of our readers and to showcase the high calibre of optometric research funded by COETF, Canadian optometry's charity.

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COETF REPORT 2012

Manitoba Association of Optometrists Museum Project

By Cheryl Bayer BSc, OD,
MAO Museum Committee Chair

In 2011, the Canadian Optometric Education Trust Fund (COETF), the Manitoba Association of Optometrists (MAO) Museum Committee, the Canadian Association of Optometrists (CAO) financially contributed to MAO's Looking Back: A Century of Vision Science display that was exhibited at the Manitoba Museum and including during the CAO Congress in Winnipeg.

The project primarily focussed on the history of optometry within the past century; however, some aspects of optics as well as the origins and designs of spectacle and contact lenses were also covered. The display opened at the Manitoba Museum on June 23, 2011 and was available for public viewing until September 5, 2011. The MAO hosted a private unveiling of the display exclusively for MAO members on June 27, 2011.

As projected, the vast majority of the artefacts displayed were borrowed from MAO members. A few artefacts, including but not limited to an old slit-lamp, prosthetic eyes and sample and model IOL's, were graciously loaned to the project by industry partners and colleagues from other specialties within the eye-care field. One of the highlights of the display was the quilt, embroidered with names of COETF donors, from a previous CAO congress in Winnipeg. The quilt provided a veritable who's who of the profession at the time.

The display itself consisted of seven covered display cases, a mock exam lane, informational poster boards (Optometry Myths, A Brief History of Optometry, The Exam Lane and The COETF Quilt). There were also two black and white poster boards displaying the interior of an optical lab circa 1920 and the outside of an optometrist's office in downtown Winnipeg. A static-mounted wall decal had the name and an explanation of how the display came into existence.

Unfortunately The Manitoba Museum does not have data available regarding the number of visitors who would have seen the display; however, they did receive a lot of positive feedback regarding its educational and aesthetic appeal. The official curators from the museum were very impressed that a group of optometrists, with the assistance of our designer, could put together such an appealing and informative exhibit! They assured me that the summer months are the busiest and that our display was ideally located for patrons to see on their way out.

The Looking Back display was also featured during Museum Bingo, an event which took place during the CAO Congress' Opening Ceremonies.

The funding provided by COETF was matched by the CAO and the additional funds required for the project were supplied by the MAO. Completion of this project on budget was possible thanks to the many long hours of volunteer work done by the MAO Museum Committee members and staff of the MAO.

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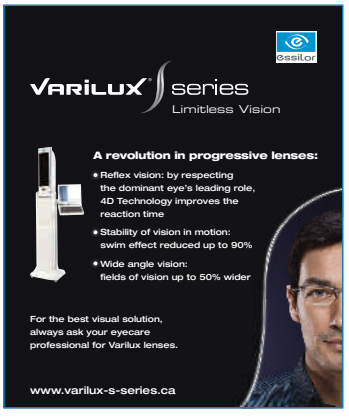
In addition, Essilor Canada is involved at all levels in the Canadian optical community. By its consumer advertising and cultural event sponsorship, it positions itself as a leader in developing the market. sponsorships, such as the Special Olympics and the « Participe pour Voir » program of La Fondation des Maladies de l'œil have been great opportunities to promote good visual health.

Essilor develops projects with all the optical schools in Canada: it collaborates very closely with the École d'Optométrie de l'Université de Montréal where thanks to an edging and mounting lab headed by one of Essilor's experts in the field, optometry students can learn about new products, the latest edging/mounting technologies as well as remote edging and electronic ordering. It has a long term partnership with the School of Optometry & Vision Science, University of Waterloo that will help enhance optometric education and further advance eye health and eye care in Canada. Essilor also brings specialized business training to eyecare professionals through programs such as the Management & Business Academy™ (MBA).

Essilor has been a sponsor of the Eye Health Council since the year 2000, supporting its activities and the diffusion of information on visual health.



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Improved ways to screen for patients with Fabry disease, involving optometry in a multidisciplinary approach

BY LANGIS MICHAUD
& CHRISTIANE AURAY-BLAIS

Introduction

Although Fabry disease has been known for more than a century (1898), this lysosomal storage disorder remains poorly recognized. With a prevalence of 1/40,000 to 1/117,000 live male births,[1] Fabry is considered one of 7,000 known rare diseases that exist in the US and reported. However, this number of patients seems to be underestimated.[3]

Many heterozygous subjects are affected without being diagnosed, no one seeing the globality of their symptoms. These are quite variable and unspecific, often leading to confusion with rheumatoid diseases or chronic inflammatory conditions.[4] In some cases, the condition remains subclinical or is not characteristic of the full spectrum of the disease. A typical patient's odyssey means multiple visits to more than ten different medical specialists before he or she achieves a confirmatory

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Disclosure: Author has received research grants and speaker fees from Genzyme Canada.

RÉSUMÉ

Objet :
La maladie de Fabry est considérée comme une maladie rare, de par sa prévalence. Cependant, ceci cache une réalité clinique toute autre en raison du nombre de cas non dépistés. Cet article vise à démontrer comment les optométristes, via un simple examen à la lampe à fente, peuvent aider à améliorer le dépistage des patients atteints, et ce, dans une perspective multi-disciplinaire.

Méthode :
Un modèle de dépistage a été instauré, en se basant sur l'éducation continue des optométristes. Les patients suspects de Fabry sont référés à l'École d'optométrie de l'Université de Montréal pour des tests supplémentaires. Dans le cas où l'histoire de cas et/ou les signes cliniques sont caractéristiques de la maladie de Fabry, un test urinaire est demandé, visant l'identification de biomarqueurs spécifiques. Si ce test s'avère positif, le sujet est alors référé à un spécialiste des maladies génétiques pour un test d'ADN et un suivi médical de sa condition.

Résultats :
Des activités de formation continue ont été réalisées à travers tout le Québec, rejoignant près de 60% des optométristes. Après 16 mois d'implantation, ce modèle a permis l'identification de 10 suspects. De ce nombre, 2 patients Fabry ont été diagnostiqués, ce qui a conduit également à l'identification de la maladie chez 5 de leurs proches. Deux autres patients, atteints de Fabry mais sans suivi médical depuis des années, ont été à nouveau pris en charge par le médecin spécialiste. À ce jour, en raison de l'implication des optométristes, 7 nouveaux patients ont donc été dépistés et diagnostiqués alors que 2 reçoivent à nouveau les soins appropriés.

Conclusion :
En se basant sur les résultats obtenus, le modèle de dépistage mis en place a été évalué comme positif. Il confirme le rôle crucial des optométristes dans le dépistage des maladies systémiques ayant des implications oculaires. L'éducation continue est essentielle à la remise à jour et en perspective de notions apprises il y a longtemps mais qui ne se rencontrent pas au quotidien dans les pratiques. De plus, ceci suggère que l'optométriste peut être impliqué dans des équipes multidisciplinaires visant le dépistage de patients à risque et de maladies, notamment celles qui entraînent des manifestations oculaires.

ABSTRACT

Purpose:

Fabry disease is considered a rare disease, based on its prevalence. It is recognized, however, that there are many individuals affected who are unscreened. This article aims to demonstrate how optometrists can help to define improved ways to screen patients affected by this rare metabolic disorder, in a multidisciplinary perspective

Methods:

A screening model, based on continuous education for optometrists was developed. Under this model, suspect patients identified by optometrists are referred to Université de Montréal's vision clinic (EOUM) for further testing and assessment. Should ocular manifestations and/or case history prove relevant to these rare diseases, a urinary test is then performed to find related biomarkers. When suspicions narrow to probable Fabry disease, the subjects are referred to metabolic disorder specialists for complete DNA testing and medical follow-up of their condition.

Results:

Continuous education lectures were given across Quebec, reaching nearly 60% of the province's optometrists. Sixteen months following the model's implementation, ten suspected patients were referred. Of these, two new Fabry patients were confirmed, leading to the diagnosis of five other relatives with the disease. Two additional persons, diagnosed as Fabry patients, but lost to medical follow-up for many years, were once again placed under the care of Fabry experts. To this point, because of optometric involvement, seven new patients of Fabry were diagnosed and two were brought back under experts care.

Conclusion:

Continuous education lectures were given across Quebec, reaching near 60% of the province's optometrists. Sixteen months following the model's implementation, ten suspected patients were referred. Of these, two new Fabry patients were confirmed, leading to the diagnosis of five other relatives with the disease. Two additional persons, diagnosed as Fabry patients, but lost to medical follow-up for many years, were once again placed under the care of Fabry experts. To this point, because of optometric involvement, seven new patients of Fabry were diagnosed and two were brought back under experts care.

Key words: *Fabry disease, screening program, corneal pigmentation, urine biomarkers, lysosomal storage disorder*

diagnosis. On average, this comes 14-16 years following the onset of the first symptoms,[5] slightly sooner for symptomatic children.[6] For Fabry patients, quality of life, is severely reduced, similar to patients with AIDS[7]. Stress, negative economic repercussions and psychological effects that can lead to moderate to severe depression can affect both patients and their relatives.[8]

Considering the life-threatening aspect of the disease, methods to improve an effective screening of the suspects, at the primary care level, are needed. This becomes essential to target specifically young male patients, more affected, before any major systemic involvement occurs. Because ocular manifestations are among the first to appear in Fabry patients, early in their life,[8] it becomes interesting to consider optometrists as key primary care players to detect and screen for Fabry disease to a greater extent. This article aims to explain how it can be done effectively, in a multidisciplinary perspective.

Fabry disease explained

Fabry disease is an X-chromosome linked disease, and counts 431 different mutations for the GLA gene.[9] It is characterized by a deficiency of the lysosomal enzyme alpha-galactosidase A, (GLA or *a*-gal A).[10] Consequently, normal degradation and catabolism processes of membrane glyco-sphingolipids, namely globotriaosylceramide (also known as GL-3, CTH, or Gb_3) can no longer be processed in nearly all cells of the human organism. GL-3 substrates cause deposits within the blood vessels. Its distribution is heterogeneous, with a preference for organs that naturally accumulate the greatest amount of it (the heart and kidneys)[11]; it also favours the vascular endothelial cells, renal dorsal root ganglion cells, the cornea and the skin[12].

Table 1: Signs and symptoms associated with the classic form of Fabry's disease

Symptoms (most appears in early childhood and adolescence)		Signs
• Acroparesthesia (numbness, tingling of the extremities) • Joint and abdominal pain • Hypohydrosis • Fever • Heat/exercice intolerance • Hearing loss and vertigo	• Gastro-intestinal disturbance or pain • Altered temperature sensitivity • Lethargy • Cefalea • Moderate to severe depression	• Angiokeratomas (bathing trunk area, ombilicus, oral mucosa, fingers, thorax) • Ocular manifestations • Facial minor dysmorphic features • Renal dysfunction • Cardiac complications • Cerebrovascular disorders (TIA, strokes)

During its course, the most severe (referred to as "classic") form of Fabry disease leads to multiple organ damage but clinical presentations typically vary from one patient to another (*Table 1*). On the other hand, milder or later-onset variants, with manifestations circumscribed to one organ, can be seen in patients showing some residual enzyme activity.[13-14]

In its classic form affecting hemizygotes, the severity of the clinical picture correlates positively with the person's age[6], but not with genotype - except where vessel tortuosity is present.[15-16]

Ocular manifestations related to Fabry disease

Ocular manifestations can be identified very early in childhood, by age three[15] or even earlier.[17] These typically occur at the same time as systemic symptoms appear, especially in hemizygous patients.

Classic ocular manifestations include vortex pigmentation of the cornea (verticillata), lens opacities (anterior whitish opacities and posterior subcapsular cataract), conjunctival vessel anomalies (tortuosity and micro-aneurysms) and retinal vessel tortuosities (*Figures 1 to 4*),[15] although such manifestations do not usually impair vision but create visual symptoms such as photophobia.

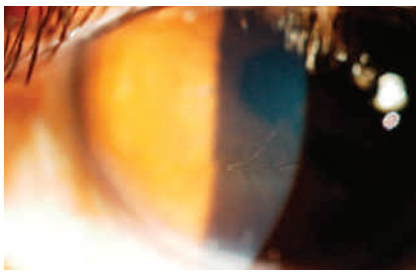

Figure 1–*Typical vortex pigmentation of the cornea (verticillata) and corneal haze surrounding the deposits.*

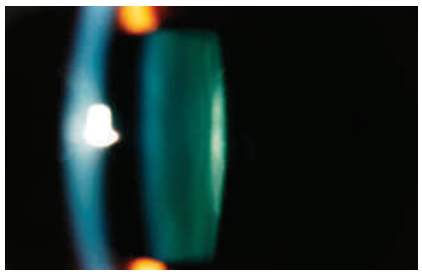

Figure 2–*Anterior whitish lens opacities and posterior subcapsular cataract in a 35 year-old Fabry patient.*

Systemic treatment

For decades, therapy was symptomatic. Since 2001 (Europe) and 2003 (USA), enzyme replacement therapy (ERT) has become an available option in the causal treatment, and been shown to be clinically beneficial.[18] This treatment helps to mitigate signs and symptoms of the disease (*Table 2*) and would potentially reduce the Gb_3 deposition that leads to irreversible organ damage.[9,19-20] ERT also provides an improvement in quality of life.[20] In addition to ERT, the most recent pharmacological approach uses genetic therapy or chemically-induced pharmacological chaperones 1-deoxygalactonojirimycin (DGJ) and galactose to stabilize the human *a*-GAL glycoprotein and consequently to increase enzyme activity within lyzozomes.[21]

Without treatment, male Fabry patients usually die 20 years earlier than the general male population, due to renal failure, progressive cardiomyopathy and/or cerebrovascular events.[22] Women also have a shorter life expectancy of 15 years compared with the general population.[22]

One way to reverse this natural course is to implement efficient screening strategies to identify suspects early in the disease process, and to refer them to Fabry specialists in a timely manner. Such a screening strategy can start with the involvement of those who see these potential patients on a daily basis. Considering that ocular manifestations are among the first to appear and the easiest to assess,[23] eye care professionals should be targeted as key players.[16]

Methods

Defining the model

In 2009, a collaborative pattern for the ocular follow-up of diagnosed Fabry patients, under the requirements of the Canadian Fabry Disease Initiative, was established between Université de Montréal, École d'optométrie (EOUM), and the genetic center of one of its university hospital (Hôpital du Sacré-Cœur de Montréal), treating most of the Fabry patients in the province. The first step in responding to CFDI's request was to review their requirements for oculo-visual examination and follow-up (time, equipment, standards, etc.) Secondly, a faculty member (LM) from EOUM was designated to take charge of the project, based on expertise in anterior segment. The third step was to secure a formal referral pathway that would function reciprocally between EOUM and the CFDI research team (under Dr. Bichet, of Hôpital du Sacré-Cœur de Montréal). The fourth step was then developed. It involved recruiting practising optometrists to screen for Fabry patients on a large scale. Several continuing education lectures were conducted across the province. During these. events, emphasis was made on the clinical course of the disease (age of onset and natural evolution), the systemic symptoms and ocular manifestations related to Fabry and the proposed screening referral pathway. Written documentation (manuscripts, posters with photos) and DVDs about Fabry and other lysosomal storage disorders were also provided as reminders for in-office use.

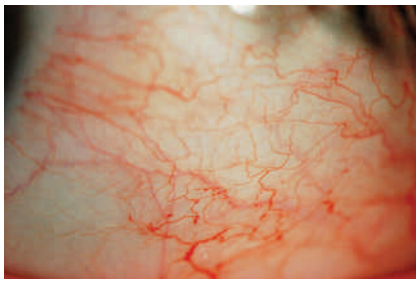

Figure 3–*Tortuosity and micro-aneurysms of blood vessels as they can be seen in the conjunctiva of a 42 year-old female patient.*

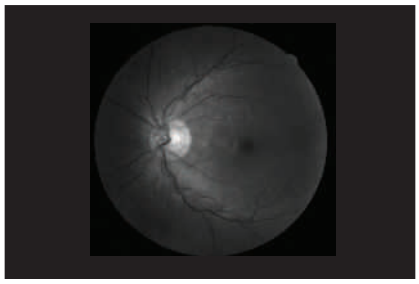

Figure 4–*Retinal vessel tortuosities, best seen in red-free photograph, in an 18 year-old Fabry patient.*

Under the developed model, a patient presenting with specific ocular manifestations and/or symptoms of Fabry is considered a suspect. The situation is explained to the patient who is offered to be

Table 2: Summary of the effects of enzyme replacement therapy (ERT) on Fabry's patients

Symptoms	ERT outcome
• Gastrointestinal disturbance /abdominal pain • Renal function (overall) • Proteinuria • Cardiac complications • Hearing • Cerebrovascular events	• Alleviated • Stabilize or slow the decline • Not improved • Beneficial- reduction in left ventricular mass (if hypertrophy present before tx) • Stabilize or improve (enzyme alpha) • No improvement if tx started after event • Unclear to prevent stroke on long term

seen at EOUM's vision clinic for confirmation and further assessment. If the clinical findings prove relevant (presence of corneal pigmentation and at least manifestation of one systemic symptom), or in the case of a positive genetic background, a urine sample collected on filter paper is sent to the CHUS Expertise Centre in Clinical Mass Spectrometry (Dr. Auray-Blais Waters' laboratory) for tandem mass spectrometry analysis for Gb_3 levels (see below). In the case of patients living outside of Montreal area, urinary test kits (filter papers and request forms) are sent to local optometrists to be administrated. Based on clinical and lab results, if enough suspicion points to Fabry (cornea verticillata, systemic symptoms and positive urinary test result for Gb_3 accumulation), a referral is made to the nearest genetic centre for complete DNA testing and follow-up.

Ocular data collected from confirmed patients are part of the CFDI registry and constitute the basis of our current longitudinal study on ocular manifestations related to Fabry.

Urinary biomarker testing

A method for screening high-risk individuals was developed to detect both male and female Fabry disease patients.[24] The methodology is reliable, efficient and specific, if done after the age of 6 years. It is based on an analysis of urinary Gb_3 using tandem mass spectrometry,[25-26] in a laboratory in Sherbrooke, QC. Urinary excretion of Gb_3 is normalized to creatinine[27] and patients are always age-matched to controls.[28] Nevertheless, it must be taken into account that patients with cardiac variant mutations who have residual enzyme activity do not excrete excessive amounts of Gb_3 in their urine.

Results

Continuous education

During the first 16 months of the project, 750 Quebec optometrists (out of 1,300 – 57.6%) participated in continuing education events, during evening and weekends.

Patient referrals

The first examination of a patient referred to the EOUM clinic was made in September 2009. Since that time, 10 patients have been seen, (*Table 3*), referred by optometrists who attended continuing education events.

The urinary test was performed on every suspect (6 out of 10) who showed signs (3 suspects) or symptoms (3 suspects) that suggested Fabry. This test

Table 3 : Demographics of the referred patients to U de M vision clinic for screening

Patient Sent by ODs	Sex	Age	*Corneal pigmentation seen*	*Positive Case History (Systemic symptoms)*	*Urinary test ordered*	*Patient sent for DNA testing*	*Fabry's confirmed*
1	F	18	Yes–typical*	Yes	Yes	Yes	Yes
2	M	22	Yes (1)	No	No	No	No
3	F	34	No	Yes	Yes	No	No
4	F	51	No	Yes	Yes	No	No
5	M	39	No	Yes	Yes	No	No
6	M	23	Yes–typical	Yes	Yes	Yes	Yes
7	F	62	Yes (2)	No	No	No	No
8	F	51	Yes-atypical	Yes(a)	Yes	Yes	No
9	M	46	Yes (3)	No	No	No	No
10	F	35	Yes (4)	No	No	No	No

- Typical means bilateral pigmentation, verticilatta type, asymetrical
- Hudson-Stahli type of pigmentation- monocular- Not related to Fabry
- Pigmentation secondary to amiodarone – symetrical OU
- Pigmentation secondary to chloroquine
- Pigmentation secondary to scarring
- Relatives affected (cousins)

allowed for the detection of 2 out of 3 suspects who showed corneal pigmentations and symptoms. The discovery of these patients led to cascade screening and the detection of other Fabry patients (5) in the family, including two young homozygous patients five and seven years old, respectively. The last patient with corneal signs had a negative result on the urinary test. However, because of her positive family background for the disease (cousins were known Fabry patients), she was referred for DNA testing which was also negative.

In addition to these patients, four additional suspects living in rural areas were co-managed with their local optometrists. Urinary tests were negative for the disease and consequently they were not referred for DNA testing.

Aside from these 19 individuals (10 suspects seen + 5 relatives + 4 co-managed patients), two other patients who had been lost to follow-up for many years were brought back under the care of either Dr. Bichet's team or a local geneticist in Quebec City, following an optometric examination.

In total, 7 new patients (2 + 5 relatives) were diagnosed as a direct consequence of the screening model we have established, and 2 more were brought back under appropriate care for their systemic condition.

Discussion

In the past, several strategies have been proposed to increase screening for patients with rare diseases.[29] These strategies have included developing and launching an interactive public website to inform and communicate with the public and with medical professionals; a mail-based survey of all primary care physicians of a known area; systematic targeted screening of the relatives of patients already diagnosed, and a newborn/population-based urine testing program should biomarkers be detected. For Fabry disease in particular, very few other initiatives have been implemented worldwide to increase the screening rate for patients. Some initiatives have attempted to look at specific groups of patients considered to be "at risk" based on their signs or symptoms. For example, studies have been conducted among hemodialysis patients, with a positive-identification success rate of 0. 2 to 1.2%.[30] A similar approach involving patients presenting with cryptogenic stroke or unexplained left ventricular hypertrophy have yielded a 3-6% success rate.[31]

In Argentina, blood sampled on filter paper (dry blood testing) from patients with signs and symptoms has been systematically sent for analysis, with a yield of detection of 4.96%. Using this approach, 70 patients were found positive for the disease within 2.5 years of implementation of the protocol.[32] One other experiment was conducted in Germany in 2003: 615 ophthalmologists were recruited and 125,908 patients were examined. Out of these, 44 subjects (3.5%) were suspects and 21(1.75%) were confirmed as Fabry patients. This result was not considered as successful as other strategies, due to the time and effort needed to screen a small number of patients.[33]

As mentioned previously, in order to increase the overall positive results from screening strategies, it is preferable to develop a targeted protocol and to constitute an interdisciplinary group to identify confirmed patients. In our case, several aspects of other screening programs were put into place with the uniqueness to include optometry in the multidisciplinary team and to be able to rely on biomarker testing at an affordable cost, compared to the higher cost of DNA testing. Referral patterns were set up considering available resources.

The innovative feature of our approach was to involve optometrists, on a large scale, as team members for the screening effort. In North America, optometrists represent the most accessible resources in primary eye care. In the province of Quebec specifically, 1,300 optometrists see 30-35% of the population every year.[34] This high level of population penetration represents a unique opportunity for screening a large population for Fabry disease.

Limitations of our analysis

We cannot estimate our rate of success as defined by the percentage of patients screened based on the total number of patients who consulted an optometrist who attended a continuing education event. We cannot estimate the male/female ratio of patients who consulted with a trained optometrist during this 16 months period of time. It is therefore difficult to compare our data with the German experiment or other screening projects.

On the other hand we can roughly approximate the value of the model. Since we know that those who attended continuing education. events represent 60% of Quebec optometrists, we can assume that they have seen as many as 2.4 million people, in theory, over the last 16 months (3 million/year/total ODs x 60% × 1.33 years). Considering that the prevalence of Fabry is 1/200,000 in the general population we can estimate that 12 Fabry patients (2.4 million × 1/200,000) should have been seen. The screening process helped to identify 7 individuals who were not diagnosed, priorly, and 2 that were lost to follow-up. In such a perspective, 9 of 12 potential patients were identified. This gives an idea of the value of the screening effort.

On the other hand, most of the patients referred to EOUM for confirmation of the disease were not found positive for Fabry, based on the lack of symptoms or due to other corneal pigmentation causes. This suggests that the educational process can be improved, or repeated. Also, sensitivity of the screening could be improved by modifying the referral criterion to only include patients with corneal deposits (unexplained by other commonly known drugs or other common causes of corneal pigmentation) and the presence of systemic symptoms or a family history.

Updated Data

The continuous education seminars were also conducted across Canada. As of December 2012, 18 other patients and relatives were found by optometrists and confirmed as Fabry patients. This brings the total of patient screened up to 25 in the last 2.5 years.

Conclusion

Overall, a year and a half after its implementation, this screening program met its goals, as it involved optometry and helped uncover new Fabry patients in Quebec.

This screening program was effective because it was based on a defined protocol, a multidisciplinary approach and a major effort to provide up-to-date information to optometrists as primary care providers.

Our ability to rely on an accessible resource, everywhere in the territory, helped immeasurably. In this sense, optometrists should be considered key players in the development of any large-scale screening program for rare diseases involving ocular manifestations.

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Under pressure: a review of normal-tension glaucoma

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Introduction

For a disease recognized as a common cause of irreversible vision loss, a universally agreed-upon definition of glaucoma remains elusive. Glaucomatous optic neuropathy (GON) is characterized by a progressive loss of retinal ganglion cells (RGC), resulting in an excavated (cupped) optic nerve head and loss of visual field sensitivity.[1] Primary open-angle glaucoma (POAG), the most common form of the disease in North America with a prevalence of 2.1%, has been described as "a multifactorial optic neuropathy characterized by acquired loss of retinal ganglion cells and optic nerve atrophy".[2] This definition has evolved over time, with specific mention of intraocular pressure (IOP) now conspicuously absent. This is at least in part in recognition of the paradox of ocular hypertension (OHT) without accompanying GON, and of GON in the presence of 'normal' IOP; it could be stated that increased IOP is sufficient, although not necessary, for the development of glaucoma. Normal-tension glaucoma (NTG) has been defined as POAG with untreated IOP within the statistically normal range of 15.5 +/-2.6mmHg; others specify that high-water IOP cannot exceed 21mmHg, at which point a diagnosis of POAG is established.[3]

Interestingly, while IOP no longer defines POAG, it does define NTG, and remains the single most important, and the only currently modifiable, risk factor in the development of glaucoma. Further, patients with NTG may demonstrate a more aggressive disease if left untreated, but often respond favourably to IOP-lowering treatment.[4] This has led investigators to suggest that the glaucoma pendulum has swung too far away from IOP, and that the disease may be best defined as the only *pressure-dependent optic neuropathy*.[5] Indeed, many recommend that the concept of distinct clinical entities be abandoned in favour of viewing glaucoma as a continuum from primarily IOP-dependent (POAG) to IOP-independent (NTG) disease.[6] Given that as many as five of every ten patients with glaucoma will present with statistically normal IOP, an understanding of the multifactorial nature of what this review will term NTG is of critical importance to the eye care practitioner.

RÉSUMÉ

En moyenne, un patient sur trois atteint de neuropathie optique glaucomateuse aura une pression intraoculaire se situant à l'intérieur des limites de la normale et recevra le diagnostic de glaucome à tension normale. Les professionnels des soins oculovisuels (et leurs patients) auront intérêt à bien connaître le diagnostic, le traitement et le pronostic de cette condition et à bien comprendre non seulement les similitudes et les différences avec le glaucome primaire à angle ouvert mais les rôles importants joués par le système nerveux central et l'état vasculaire systémique.

Mots clés : *Glaucome à tension normale (GTN), glaucome primaire à angle ouvert (GPAO), hystérèse cornéenne (HC), hémorragie discale (HD), atrophie péripapillaire de la zone bêta (APPβ), pression de perfusion oculaire (PPO), dysrégulation vasculaire, pression du liquide céphalorachidien (PLCR), différence de pression à travers la lame criblée, neuroprotection*

Epidemiology and Risk Factors

Even more than POAG, NTG tends to be a disease of the elderly, with a prevalence of 1.6% in the population over the age of 75; up to 30% of patients with NTG, however, will be under the age of 50.[7] Upon diagnosis, the rate of progression and response to treatment appear unrelated to age.[8] There is evidence that NTG is more common, more severe, and more resistant to treatment in females.[9,10] There also appears to be an ethnic predilection, as upwards of 90% of Japanese and Mongolian patients with POAG present with IOP less than 21mmHg; Caucasians, however, tend to manifest more serious disease.[11-13] A family history of glaucoma is reported by 30 to 40% of patients with NTG. Investigators have observed that patients with NTG tend to be of lower body weight and body-mass

index (BMI).[14] It has been hypothesized that patients with NTG tend to be more health-conscious (in fact, some would suggest health-anxious), and exhibit more proactive health behaviour. Myopic patients may demonstrate progressive GON in the presence of low IOP, and tend to have difficult to interpret, often tilted, optic nerve heads.[15,16] While a discussion of genetics is beyond the scope of this review, upwards of twenty genes associated with POAG have been identified, and there is evidence that several may be specific for NTG. At least two gene loci are associated with NTG and exfoliative glaucoma; these loci influence transforming growth factor beta (TGF-β), perhaps suggesting a future neuroprotective target.[17,18]

Pathophysiology – *Under Pressure*

Although by definition NTG presents with IOP within the statistically normal range, admittedly an arbitrary construct with no pathophysiologic meaning, further reducing pressure tends to slow disease progression, albeit not universally.[19] Nocturnal IOP elevation, particularly in concert with nocturnal systemic hypotension, is very significant; sleep lab and telemetric studies demonstrate that as many as two out of every three patients exhibit maximal IOP outside regular office hours.[20-22] In recognition of the impact of corneal biomechanical properties on applanation tonometry (AT), and potentially on ocular integrity itself, these properties have recently received greater attention. Patients with NTG tend to have central corneal thicknesses (CCT) approximately 30 microns below the population mean of 550 microns, leading some to hypothesize that a subset of patients with POAG are misdiagnosed with NTG.[23,24] It has been proposed that the increased prevalence of NTG among some ethnic groups (individuals of Japanese and African descent) may be partly attributable to thin CCT.[25,26] Interestingly, reduced CCT was more common in patients with NTG and vascular dysregulation than in those without, suggesting more than simply an underestimation of IOP.[27] While the Ocular Hypertension Treatment Study (OHTS) did lead to fewer patients with OHT and more patients with 'normal' pressures being treated, the association between CCT and glaucoma, specifically whether CCT may be considered a proxy for ONH biomechanical integrity, remains unclear.[28] Recently, the role of corneal hysteresis (CH), reflecting the cornea's viscoelastic ability to dampen fluctuations in IOP and reduce optic nerve head (ONH) strain, has received attention as another potentially important biomechanical parameter.[29,30] While influenced by CCT, lower CH is consistently and independently associated with an increased risk of GON.[31] There is evidence that a related parameter, corneal resistance factor (CRF, a measure of ocular rigidity), is similarly reduced in cases of concurrently low but fluctuating IOP – that is, NTG.[32] Whereas attempts to 'correct' IOP for CCT alone have proven ineffective, 'corneal compensated IOP (IOP_{cc})', encompassing a more global corneal biomechanical analysis, may hold promise: IOP_{cc} was essentially equal to AT in POAG, but significantly higher in NTG.[33] Whether reduced CH and CRF are risk factors for, or a result of glaucoma, and whether they will prove to be better proxies for ONH biomechanical integrity than CCT alone is yet to be determined; further study is necessary.[34]

Reduced ocular perfusion is found in the majority of patients with glaucoma, more so in the presence of NTG than POAG.[35] Cardiovascular disease, including increased blood viscosity, diabetes,

ABSTRACT

On average, every third patient with glaucomatous optic neuropathy will present with intraocular pressure within the statistically normal range, manifesting normal-tension glaucoma. Eye care practitioners (and their patients) will benefit from a familiarity with the diagnosis, treatment, and prognosis of this condition, including similarities to, and differences from, primary open-angle glaucoma, and the important roles played by the central nervous system and systemic vascular status.

Key words: *normal-tension glaucoma (NTG), primary open-angle glaucoma (POAG), corneal hysteresis (CH), disc hemorrhage (DH), beta-zone peripapillary atrophy (βPPA), ocular perfusion pressure (OPP), vascular dysregulation, cerebrospinal fluid pressure (CSFP), trans-lamina cribrosa pressure differential, neuroprotection*

and both systemic hypertension and hypotension, has been identified as a risk factor for the development of glaucoma, and may be predictive of a poor response to treatment.[36,37] In fact, patients tend to show increased risk of glaucoma at both extremes of blood pressure (BP), albeit more so with hypotension, which results in generalized poor perfusion. Hypertension leads to atherosclerosis, damaging endothelial cells and impairing autoregulation, rendering the ONH more susceptible to decreased vascular perfusion, increased IOP, and metabolic demands.[38] In the Collaborative Normal-Tension Glaucoma Study (CNTGS), patients *without* cardiovascular disease tended to progress rapidly when untreated, but benefitted from IOP reduction; vasospastic disease was more predictive of progression than occlusive disease. Magnetic resonance imaging (MRI) of the brain has demonstrated vascular insufficiency in patients with NTG, while cardiac studies have reported an increased incidence of silent myocardial infarction.[8,38] In patients with low IOP who show progressive visual field (VF) and ONH damage, systemic hypotension causing low ocular perfusion pressure (OPP, a surrogate being the difference between brachial BP and IOP) may undermine the benefits of low IOP.[39-41] The risk of GON increases as much as six-fold in the presence of low OPP; a diastolic OPP of less than 55mmHg has been associated with a doubling of relative risk.[42,43] A physiologic nocturnal BP dip secondary to reduced sympathetic nervous system activity that coincides with a nocturnal IOP spike can cause a pronounced OPP trough.[44,45] Patients with nocturnal BP dips of greater than 10 to 15% demonstrate more significant retinal nerve fiber layer (RNFL) and VF loss.[46,47] Some patients may experience iatrogenic systemic hypotension secondary to aggressive treatment of systemic hypertension.[48,49] Indeed, aggressive lowering of BP has been shown to increase ONH cupping in patients *without* glaucoma. Significant variations in OPP, like IOP, may be an independent risk factor for GON and VF deterioration within ten degrees of fixation.[50-52] OPP may be increased by lowering IOP and avoiding overtreatment of systemic hypertension (of course, deliberately elevating BP increases comorbidities), and its variability reduced by smoothing IOP spikes and BP troughs.[53]

Patients with NTG often have histories of tinnitus, migraine headache, and Raynaud's phenomenon, all manifestations of primary vasospastic vascular dysregulation, an imbalance between autoregulatory vasoconstrictor and vasodilator stimuli.[4,14] Patients with migraine, especially women, seem particularly predisposed to rapid (2.6×) progression of NTG, and lowering IOP in women with migraine may be less protective than in those without.[8,12,54] Vasospastic disease is more common in women, particularly post-menopause, and in patients of Japanese descent, two populations known to be at higher risk of NTG. Hemorrhaging within the fingernail capillary bed, an accepted sign of vascular dysregulation, is statistically more common in patients with glaucoma, particularly in those with a history of disc hemorrhage, and may be a helpful ancillary indication of vascular insufficiency.[55] Reduced arterial and peripapillary retinal capillary blood flow has been demonstrated in patients with NTG; many of these patients exhibit vasospastic tendencies and asymmetric VF loss that correlates to interocular asymmetries in blood flow and velocity.[56,57] Episodic vasospasm and rebound hyperperfusion can lead to local inflammation and oxidative damage.[35] Some patients with presumed GON and statistically normal IOP will have a history of hemodynamic crisis (sudden and severe systemic hypotension); such patients tend to show minimal if any progression over time.[36,37] In fact, in an early study, Drance noted that nearly 90% of patients with NTG had experienced transient or sustained systemic hypoperfusion.[39]

While eye care practitioners routinely measure the trans-corneal pressure differential (the difference between IOP and atmospheric pressure), what truly influences the ONH through disruption of RGC axoplasmic flow is the translamina cribrosa pressure differential (the difference between IOP and orbital cerebrospinal fluid pressure [CSFP]).[58,59] The elevated translamina cribrosa pressure differential of POAG caused by high IOP may be mimicked in NTG by a low CSFP within the optic nerve subarachnoid space (ON SAS).[60] CSFP

is lower in patients with NTG than in patients with POAG; both groups exhibit lower CSFP than controls (the average being between 5 and 15mmHg), who, in turn, exhibit lower CSFP than patients with OHT.[61,62] The inter-group CSFP differences appear similar to the inter-group IOP differences observed in other studies.[63] Low CSFP and high trans-lamina cribrosa pressure differential are both positively correlated with GON and glaucomatous VF loss.[64] The thinning of the lamina known to occur in GON may exacerbate the trans-laminar pressure differential. Given that pulsatile mechanical stress is more damaging than steady, the role of CSFP fluctuation, akin to IOP fluctuation, is also receiving attention.[65] In patients with NTG, the density of CSF in the ON SAS is significantly lower than intracranial CSF; this impairs fluid exchange and leads to relative CSF stagnation within the ON SAS, with potentially detrimental impact upon RGC axons.[66] All three pressures (IOP, OPP, and CSFP) are independent yet interrelated, and may be simultaneously influenced by an as yet undetermined systemic mechanism.[67] Indeed, one cannot discount the possibility that GON and VF loss attributed to low OPP is actually secondary to low CSFP, as the latter is often found in the presence of systemic hypotension. Neuroimaging has demonstrated a narrower ON SAS width in patients with NTG, suggesting lower CSFP in that space.[68] Given that direct CSFP measurement through lumbar puncture (LP) is invasive and not without risk, such a surrogate noninvasive means of assessment would certainly be of value.

Structural Change

Some investigators feel that NTG exhibits an extreme amount of ONH cupping, typified by a pale, gently sloping, moth-eaten appearance, with broad thinning of the inferior temporal aspect of the neuroretinal rim (NRR).[69] Others suggest that the disc changes in NTG represent localized areas of nonperfusion (a focal ischemic glaucoma), preceding or coinciding with adjacent wedge or slit RNFL loss that results in initial severe VF loss that is very close to fixation.[70] This type of damage appears more common in female patients with a history of systemic vasospasm and migraine.[71] Subsequent confocal scanning laser ophthalmoscopic (SLO) studies, however, found no significant differences in optic disc topography in cases of POAG and NTG.[72] The rate of progressive ONH damage may be greater in patients with NTG than in those with POAG, particularly in patients with already-advanced GON, where lowering IOP may be of marginal benefit.[73]

First described by Bjerrum over a century ago, rising to prominence through the work of Drance some sixty years later, the etiology of disc hemorrhages (DH) remains unclear. Rather than arguing cause versus effect (primary infarction versus secondary degeneration), a mixed-mechanism theory is gaining traction.[74-77] These small pre-laminar radial flame- or splinter-shaped hemorrhages occur most commonly at the inferior temporal aspect of the ONH, adjacent to areas of focal NRR thinning and RNFL loss, and within two clock hours of areas of beta-zone peripapillary atrophy.[78-82] They are two- to five-fold more common in patients with NTG than in those with POAG or OHT, or without glaucoma. Indeed, 15 to 42% of patients with NTG demonstrate DH at baseline or follow-up, versus 7 to 37% of patients with POAG, 8% of those with OHT, and only 0.2 to 0.5% of the non-glaucomatous population.[83-88] DH are found frequently in older patients with systemic hypertension, in patients with vasospastic disease, and in women with a history of migraine.[89,90] They are more common in the presence of IOP instability, and relatively rare in patients with secondary OAG, who typically present with significant IOP elevation. In the Early Manifest Glaucoma Trial (EMGT), over half the participants demonstrated DH at least once over an average of eight years; most will be found within the first three to five years of diagnosis.[91] However, given that the prevalence of glaucoma is 2 to 4%, up to 70% of isolated DH will be found in patients not (yet) diagnosed with the disease.[83] DH are best detected through photography: being transient and subtle, they are overlooked during clinical exam as often as 84% of the time. Concurrent disease processes, including posterior vitreous detachment, diabetes, or venous occlusion, must be considered in the differential diagnosis.

DH have long been considered a strong and independent risk factor

for progressive GON, increasing the hazard rate by a factor of four to six, more so in patients with NTG than POAG, particularly in elderly patients with pre-existing VF loss.[92-98] In patients with OHT, DH were strong indicators of future conversion to POAG, and were up to five times more common following conversion.[94] In the CNTGS, DH was considered a reason to initiate or augment therapy, and was a strong predictor of more rapid progression (2.7×) of untreated NTG.[99] Up to two-thirds of VF and three-quarters of ONH show progressive change following DH; VF loss may occur at two to eight times the rate, particularly when DH are inferior temporal and/or multiple.[74,77,81,90,94,98] Eyes with DH were up to fourteen times more likely to have a worsening of RNFL status within one year. RNFL, NRR, and VF loss can also precede DH by weeks or months; retrospective evaluation has indicated that all eyes developing DH show evidence of preexisting NRR notching.[100] Eyes with enlarging RNFL defects are four times more likely to demonstrate DH, with 80% occurring at the border between unhealthy and healthy RNFL, suggesting that this is the most active anatomical site of glaucoma progression. Such RNFL defects enlarge toward the fovea nearly 90% of the time, causing more central VF change. In light of these relationships, some investigators now consider DH a sign of, rather than a risk factor for, progression.[101] DH become less common in end-stage glaucoma, and then are found nasally, adjacent to the only remaining viable NRR and peripapillary vasculature.[90,95] Once DH is detected, careful documentation and vigilant follow-up is critical; many investigators suggest every few months, given that the average duration of DH is eight to ten weeks. Recurrent bleeds, often within two years and two clock hours of the initial DH, are found in up to 73% of patients with NTG; eyes that re-bleed tend to have a significantly lower IOP than eyes with isolated DH.[76,77,87] A number of studies suggest that patients with recurrent DH have a higher probability of progressive GON, RNFL loss, and more rapid rates of VF deterioration.[102,103] As a rule, patients with DH do not respond as well to treatment as those without.[12] In fact, moderate IOP lowering may not alter the rate of DH, indicating a less IOP-dependent form of glaucoma requiring more aggressive pressure reduction even in the presence of what would otherwise be considered well-controlled IOP.[104]

Beta-zone peripapillary atrophy (βPPA) is an absence of RPE and thinning of Bruch's membrane and the choriocapillaris immediately adjacent to the ONH; alpha-zone PPA is pigment irregularity just peripheral to the beta-zone when the latter is present. From a semantic perspective, some argue that the term *parapapillary* is more correct that *peripapillary*, as the atrophy may not completely encircle the ONH. While present in 15 to 20% of normal eyes, βPPA has been noted to be larger and more frequent in eyes with glaucoma, and is considered an independent, location-specific, and severity-dependent risk factor for the progression of GON.[105-108] Many believe βPPA to be more common in NTG, particularly in younger patients with moderate to severe disease.[109-111] Other investigators feel that βPPA in NTG does not differ from that in POAG, but still helps differentiate NTG from non-glaucomatous optic neuropathy.[112] Nasal βPPA is present in only 1 to 9% of normal eyes, but 15 to 71% of glaucomatous eyes; this may also aid in differential diagnosis.[113-115] Assessing βPPA stability may be particularly valuable in the evaluation of small ONH in which intrapapillary glaucomatous damage can be more difficult to detect.[116] Conversely, βPPA may be less helpful in the evaluation of oblique or highly myopic ONH and in patients of Asian ethnicity, where peripapillary alterations are more prevalent to begin with; ironically, patients with NTG are commonly Asian and/or myopic.[117] βPPA is often found adjacent to an area of focal NRR loss and/or DH, and large areas of βPPA are predictive of future DH. Interestingly, βPPA and DH are associated even in the absence of glaucoma, suggesting a shared etiology of local vascular insufficiency and breakdown of the blood-retina barrier.[115] Some hypothesize that a disturbance of ONH perfusion *secondary* to βPPA may result in sectoral ischemia, or that leakage of vasoactive substances through compromised peripapillary vessels can damage the RNFL in the face of normal IOP.[109] In these cases, βPPA is felt to be a

risk factor for, rather than a sequelae of, glaucoma. That being said, βPPA is not necessarily static; progression can be seen over time, three to five times more commonly in patients with glaucoma, associated with increasing GON and VF loss.[110] The presence and enlargement of βPPA shows significant correlation with RNFL thickness and rate of thinning (particularly in the inferior quadrant), cup/disc ratio, mean VF loss, and NRR area.[114] βPPA shows a strong correlation with VF defects within five degrees of fixation known to be more common in NTG. Both the absolute scotoma of βPPA and the relative scotoma of alpha-zone PPA will cause an enlarged blind spot. βPPA can be detected and monitored qualitatively through ophthalmoscopy and photography, quantitatively through imaging techniques including SLO and optical coherence tomography (OCT).

Functional Change

As already noted, as many as two-thirds of cases of NTG present with initial VF defects that threaten fixation; these are strong predictors of future VF deterioration and visual acuity loss.[118] VF defects that threaten fixation are best monitored with both 24- or 30-degree and 10-degree testing strategies. Significant VF deterioration appears to occur in one-sixth to one-third of patients with treated NTG.[93] That being said, recall that the CNTGS showed that over half the patients with untreated NTG manifest no discernible deterioration over five to seven years. While conventional wisdom holds that most cases progress slowly, there is significant variability in rates of progression, even more so than in POAG: a ten-fold range from 0.2 to 2.0dB per year.[99] More VF loss is seen in NTG with higher IOP, but IOP variability over both short- and long-term appears to be an important predictor of, and perhaps independent risk factor for, glaucomatous VF progression, particularly in cases of low IOP.[119] The challenge, in both NTG and POAG, is to identify those at risk of rapid progression, and initiate early and aggressive treatment. Particular attention must be paid to localized VF progression, which has been proven to be a strong predictor of future DH, and focal GON.[74] It has long been reported that thinning of the RNFL, documented through both qualitative and quantitative means, is an early sign of GON, often preceding VF loss.[120-122] Spectral domain OCT (SD OCT) has indicated that RNFL thinning is most significant at the superior and inferior temporal aspects of the ONH, and correlates strongly with VF deterioration.[123] It has been proposed that loss of 17 to 20% of age-matched average RNFL thickness, to a level of 70 to 75 microns, is the 'tipping point' for structural change, whereas as many as half the RGC may need to be lost to manifest functional (VF) change.[124,125] Given that RNFL thickness assessed through OCT demonstrates a floor effect at approximately 50 microns, it may be best to monitor early GON through structural analysis, but advanced GON through functional measures.[126] Ideally, a combined index of structure and function would allow better detection, prediction, and follow-up at any stage of the disease continuum than either parameter in isolation.[127]

Management

Given that IOP remains important in the pathogenesis of NTG, the use of topical anti-glaucoma drugs remains the mainstay of treatment.[128] The CNTGS demonstrated that lowering IOP by 30% from baseline, to an average of 11mmHg, reduced the risk of progression nearly three-fold.[19] That being said, 65% of untreated eyes showed no progression over five years of follow-up, while up to 20% of treated eyes did.[8] The EMGT, a study in which over 50% of the cohort had NTG, indicated that reducing IOP halved the risk of glaucomatous damage, most significantly in the face of already-low pressures.[129,130] The conclusion that each 1mmHg IOP reduction reduced the risk of glaucoma damage by 10% emphasized the importance of vigilant monitoring, and that 'last millimeter of mercury of effect'.[107] This aggressiveness must be tempered, however, by the realization that glaucoma treatment is likely to continue for the duration of the patient's life; the side effects of medicine and surgery on quality of life must be carefully considered.[131-134] Lowering peak and mean IOP and blunting IOP fluctuation decreases the risk and rate of glaucomatous VF loss.[135] While dealing with an admittedly different population, the Advanced Glaucoma

Intervention Study (AGIS) indicated that patients with IOP consistently below 18mmHg demonstrated little if any VF progression over six years; even occasional elevations above 18mmHg resulted in more VF loss.[135] Strict adherence to an individualized and appropriate target IOP appears to result in better VF preservation.[132] Particularly with NTG, clinicians must realize that in-office IOP assessment is but a moment in time, and that structural and functional damage may occur exponentially with undetected IOP spikes. This makes the goals of lowering mean and peak IOP, and smoothing short- and long-term fluctuations, equally critical. In the presence of extreme GON, the disease may become essentially pressure-independent, emphasizing the importance of early and effective intervention.

A review of clinical trials indicates that latanoprost, bimatoprost, timolol, and brimonidine are effective in reducing IOP in patients with NTG: latanoprost seems most effective in reducing trough IOP and smoothing the diurnal curve, while brimonidine is most effective in reducing peak IOP, but least effective at trough.[136] The World Glaucoma Association recognizes topical carbonic anhydrase inhibitors (CAI) as having a beneficial effect on ONH perfusion through increasing blood flow velocity in the short posterior ciliary arteries (SPCA); prostaglandin analogs (PA) appear to be hemodynamically neutral.[137] PA and CAI lower both diurnal and nocturnal IOP, whereas beta-blockers are ineffective during the nocturnal period. Among the beta-blockers, betaxolol may lower vascular resistance more than timolol, leading to better VF preservation despite higher treated IOP. That being said, should treatment of NTG be initiated, an aggressively low target IOP (approaching episcleral venous pressure of approximately 10mmHg) may be preferable; this target may require multiple medications or the consideration of surgery.[19] In addition to traditional topical management, it has been suggested that systemic calcium channel blockers may be protective in cases of NTG through reduction of vasospasm; others argue against their use due to the potential for nocturnal systemic hypotension and reduced OPP.[15,44,73,104] Systemic CAI may concurrently lower both IOP and CSFP, leaving the trans-lamina cribrosa pressure differential unchanged, providing little benefit in the management of chronic glaucoma.[61]

As an adjunct, moderate aerobic exercise may be beneficial in both stabilizing the cardiovascular system and reducing IOP.[138]

Neuroprotection is defined as a therapeutic paradigm for slowing or preventing death of neurons (in the case of glaucoma, RGC and their axons) in order to maintain their physiologic function.[139] Whether secondary to excitotoxic neurotransmitters (glutamate), ischemic/oxidative injury and subsequent reperfusion inflammation, blockage of growth factors/neurotrophins, mitochondrial dysfunction, or some other mechanism, apoptosis (programmed cell death) may continue independent of the level of IOP.[140] Glaucomatous damage is not limited to the ONH; alterations in the visual pathway behind the globe (including lateral geniculate nucleus and visual cortex) have been noted in the absence of detectable RGC loss.[141] Given that current treatments are limited to IOP-lowering, yet some patients with glaucoma continue to progress despite low pressures, a treatment that is independent of IOP is certainly enticing. Some current glaucoma medications appear to have neuroprotective activity: in the Low-Pressure Glaucoma Treatment Study (LoGTS), brimonidine demonstrated a beneficial effect on VF preservation independent of IOP-lowering; as previously noted, dorzolamide has been proven to increase OPP.[49,137] Memantine and bis(7)-tacrine (glutamate modifiers used in the treatment of Alzheimer's disease, a disease that may share some basic mechanisms of cell death with glaucoma) are among a growing number of systemic agents being studied.[142,143]

In situations where there are atypical clinical findings (age less than 50, visual acuity less than 20/40, ONH pallor, vertically aligned/neurologic VF defects, lack of correlation between structural and functional change, and/or progression at very low IOP) neuroimaging of patients with NTG to rule out compressive lesions of the optic nerve has been suggested.[16]

Conclusion

Normal-tension glaucoma is an increasingly common, and certainly challenging, clinical presentation. The challenge begins with differential diagnosis, and continues through follow-up. Practitioners must gather, integrate, and interpret a myriad of data: ophthalmic and systemic, past and present. Given that many untreated patients show little progression over time, careful observation prior to initiating therapy is certainly prudent. That being said, it may be wise to consider more aggressive treatment of NTG in patients with multiple risk factors – for example, a young female with a history of migraine presenting with a disc hemorrhage. While the mainstay of contemporary management remains topical IOP-lowering, the recognition of ocular perfusion and cerebrospinal fluid pressure as important contributing factors may lead to their modification becoming part of the treatment paradigm.

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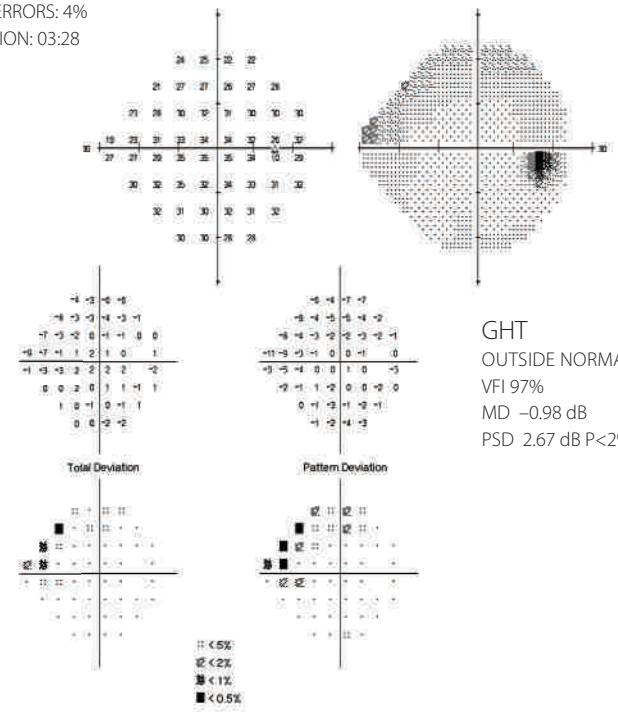
Appendix – Illustrative Case Presentation

RIGHT EYE

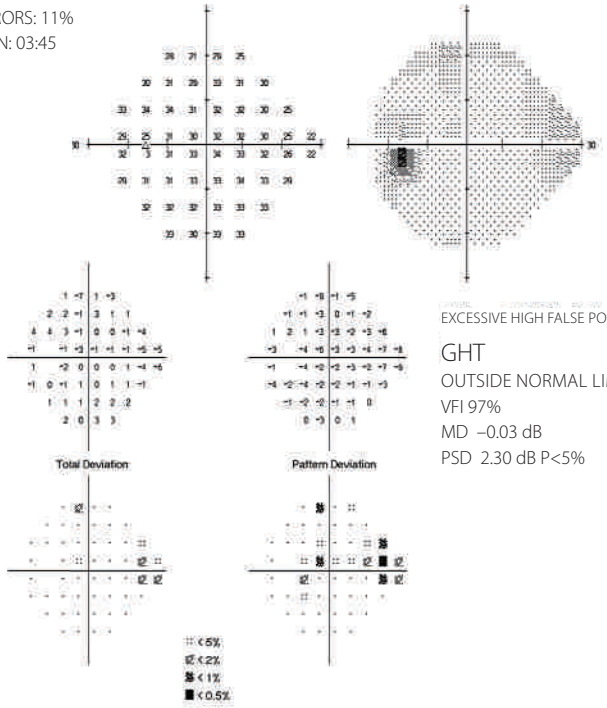
FIXATION LOSSES: 0/13
FALSE POS ERRORS: 9%
FALSE NEG ERRORS: 4%
TEST DURATION: 03:28



GHT
OUTSIDE NORMAL LIMITS
VFI 97%
MD –0.98 dB
PSD 2.67 dB P<2%

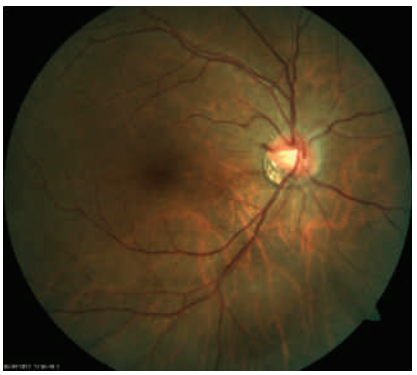
LEFT EYE

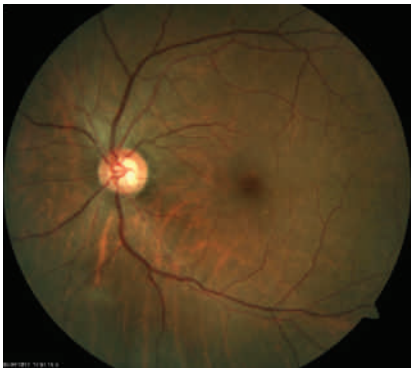
FIXATION LOSSES: 5/12 xx
FALSE POS ERRORS: 18% xx
FALSE NEG ERRORS: 11%
TEST DURATION: 03:45



EXCESSIVE HIGH FALSE POSITIVES***

GHT
OUTSIDE NORMAL LIMITS
VFI 97%
MD –0.03 dB
PSD 2.30 dB P<5%





This case encapsulates the diagnostic dilemma of NTG.

The patient in question is a 51-year old myopic (-7.00D) Asian female who discontinued treatment with prostaglandin analog two years ago. She takes no systemic medications, and denies any symptoms of systemic vascular dysregulation. Her IOPs are 14 and 15mmHg; her CCTs are 494 and 493 microns.

The right ONH (top photo) is obliquely inserted, with superior temporal DH, inferior temporal βPPA, and adjacent RNFL defect. The left ONH shows inferior temporal NRR thinning with adjacent RNFL defect. Initial VF analysis (albeit with questionable reliability; confirmation pending) shows an early superior nasal step in both right and left. Her GP is being consulted to ensure that her systemic vascular status is satisfactory. Pending confirmatory VF analyses, topical treatment with prostaglandin analog (with a target pressure approaching the episcleral venous pressure of ~10mmHg) is likely to be initiated.

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