CANADIAN JOURNAL OF OPTOMETRY REVUE CANADIENNE D'OPTOMÉTRIE

CJO RCO

VOL. 54 NO. 4 WINTER / HIVER 1992/93

Vision Awareness Week, March 1–7, 1993

La Semaine de la vision, 1–7 mars, 1993

Even Better News!

FreshPak is available in two new environmentally responsible formats.

Now your patients can enjoy all the benefits of FreshPak and feel good about reducing the impact on the environment.

Using today's advanced technology, we've created our new packages from recycled materials and vegetable-based inks. The smaller version, EnviroPak, is the new replacement kit. The larger unit replaces our current packaging. It retains the same consumer appeal for new users, with a minimum of packaging. Increasingly, your patients want environmentally responsible manufacturing. And so do we.

Now you can choose the FreshPak you want, knowing you are doing your part. For more information call your Bausch & Lomb representative at 1-800-668-7510.

BAUSCH & LOMB
Healthcare and Optics Worldwide

FreshPak and Bausch & Lomb are trademarks of Bausch & Lomb Incorporated.

Canadian Journal of Optometry • la Revue canadienne d'optométrie
Vol. 54, No. 4
Winter / hiver 1992/93
(Date of issue: Feb 1993)
(Date de parution : fev 1993)
ISSN 0045-5075

The Canadian Journal of Optometry (CJO*RCO) is the official publication of the Canadian Association of Optometrists / **La Revue canadienne d'optométrie (CJO*RCO)** est la publication officielle de l'Association canadienne des optométristes: Suite 301 — 1785 Alta Vista Drive, Ottawa, Ontario, K1G 3Y6. (613) 738-4412; Fax. / Télécopieur (613) 738-7161. Publications Mail Registration No. 5807 / Envoi de publication — Enregistrement no. 5807.

•

The Canadian Journal of Optometry • la Revue canadienne d'optométrie (CJO*RCO) is the official publication of the Canadian Association of Optometrists (CAO). However, opinions and commentaries published in the **CJO*RCO** are not necessarily either the official opinion or policy of the CAO unless specifically identified as such.

The Canadian Journal of Optometry • la Revue canadienne d'optométrie (CJO*RCO) welcomes new advertisers. In keeping with the **CJO*RCO**'s goal of advancing the awareness, education and professionalism of the members of the Canadian Association of Optometrists (CAO), any and all advertising material may be submitted, prior to its publication, for review by the National Publications Committee of CAO. CAO reserves the right to accept or reject any advertisement submitted for placement in the **CJO*RCO**.

•

The Canadian Journal of Optometry • la Revue canadienne d'optométrie (CJO*RCO) est la publication officielle de l'Association canadienne des optométristes (ACO). Les avis et les commentaires publiés dans la **CJO*RCO** ne représentent toutefois pas nécessairement la position ou la politique officielles de l'ACO, à moins qu'il en soit précisé ainsi.

The Canadian Journal of Optometry • la Revue canadienne d'optométrie (CJO*RCO) est prête à accueillir de nouveaux annonceurs. Dans l'esprit de l'objectif de la **CJO*RCO** visant à favoriser la sensibilisation, la formation et le professionnalisme des membres de l'Association canadienne des optométristes (ACO), on pourra soumettre tout matériel publicitaire avant publication pour examen par le Comité national des publications de l'ACO. L'ACO se réserve le droit d'accepter ou de refuser toute publicité dont on a demandé l'insertion dans la **CJO*RCO**.

•

Chairman, National Publications Committee / Président, Comité national des publications
Dr Pasq Marcantonio

Senior Editor / Rédacteur en chef
Dr Mitchell J Samek

Academic Editors / Rédacteurs académiques
University of Waterloo: Dr B Ralph Chou
Université de Montréal : Dr Pierre Simonet

Managing Editor / Rédactrice administrative
Heather L Ebbs

CAO Director of Communications / Directeur des communications de l'ACO
Michael J DiCola

Printing / Impression
Love Printing Service Ltd

Translation / Traduction
Tessier Translations / Les Traductions Tessier

Cover / Couverture
Viston safety
La sécurité de la vision

CJO•RCO

Editorial / Éditorial

Editorial: Once again!
MJ Samek 172

Éditorial : Encore une fois!
MJ Samek 172

President's Podium: Optometry's celebration of 25 years at Waterloo
MR Hansen des Groseilliers 175

Mot du président : L'optométrie célèbre ses 25 ans à Waterloo
MR Hansen des Groseilliers 175

Articles / Articles

Corneal dystrophies and degenerations
Part 2: The stroma
M Braun, WM Lyle 180

How useful is the VisTech MCT8000 for contrast sensitivity and glare testing?
DB Elliott 194

Clinical / Clinique

Clinical Diagnosis: *C Chiarelli* 177

Case Reports: Acquired sixth nerve palsy
M Marconi 200

Clinical Comment: Chemical soaking solutions versus hydrogen peroxide for soft lens disinfection
D Fonn, S Grunert 202

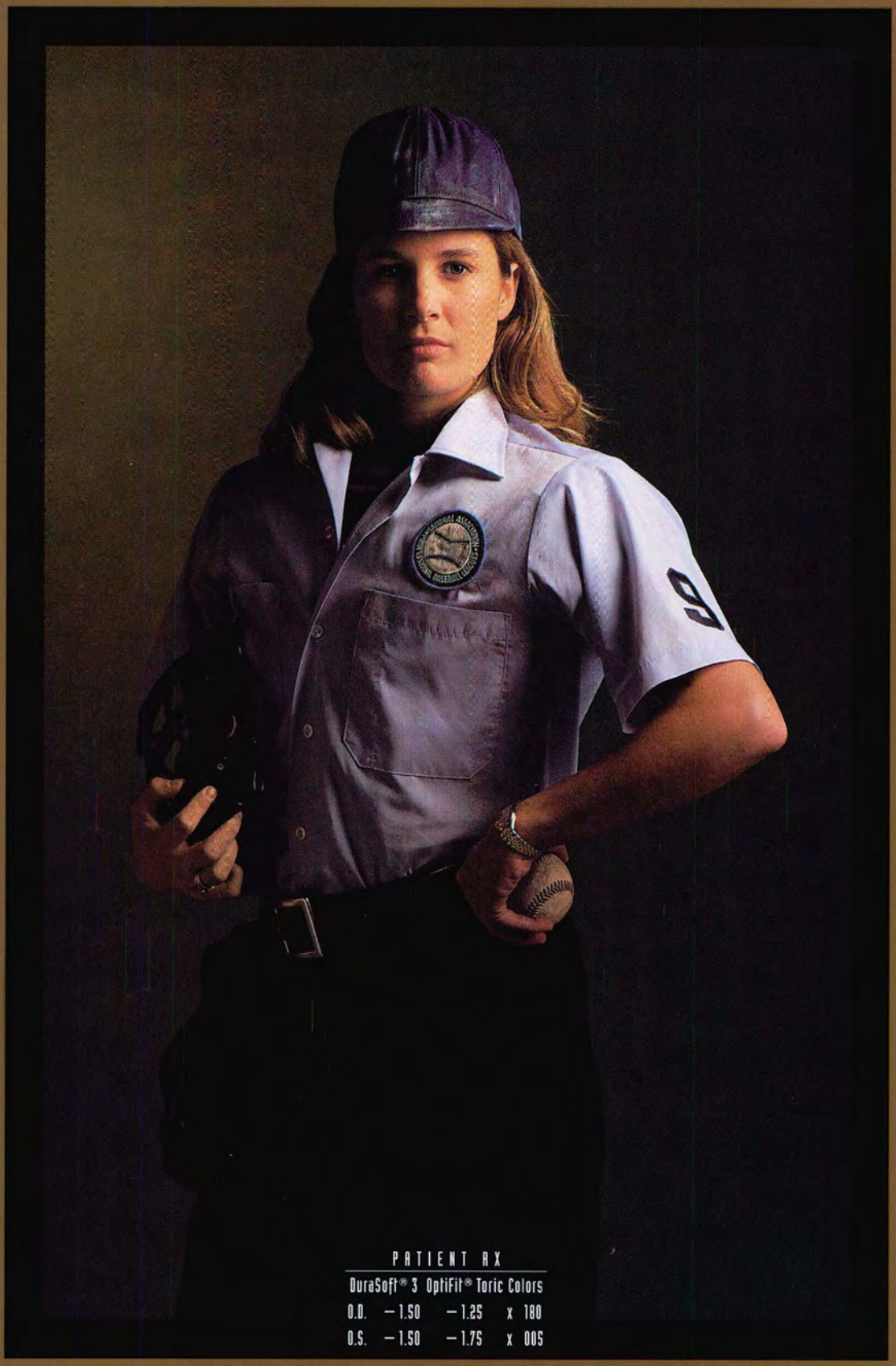
Features / Rubriques

Annual index / Index annuel 208

Financial Foresight / Prévisions financières
Robert Décarie 214

Vision Care News / Actualité oculo-visuelle 220

Calendar / Calendrier 228



PATIENT RX

DuraSoft® 3 OptiFit® Toric Colors

O.D. −1.50 −1.25 x 180

O.S. −1.50 −1.75 x 005

OPTIFIT® CALLS 140 GAMES A YEAR.

Minor league umpire Eileen Winn needs a good eye. In fact, she needs two. So when it comes to her contact lenses, there's no room for error. Which is why her optometrist outfitted her with DuraSoft® OptiFit® Torics from Wesley-Jessen. *Eileen's doctor knows that OptiFit lenses offer the widest range of stock parameters available, and that they're manufactured with exacting precision.* OptiFit Torics are cut on an air-suspended, vibration-free computerized lathe to deliver superior visual acuity, consistent reproducibility, and a lens that fits like a glove. Their back-surface design ensures stability. And the unique Opti-Mark® orientation system assists Eileen's doctor in determining lens rotation, and helps Eileen get her lenses on right every time. Which allows Eileen more time on the field and far less time in her optometrist's chair. So whether your patients are calling strikes or calling on clients, you can be sure they'll get all-star performance from their OptiFit lenses. No matter what their game.

DURASOFT® OPTIFIT® TORICS

VISION WITH PRECISION.

Wesley-Jessen® CORPORATION

©1993 Wesley-Jessen Corporation WESTR9303

Editorial

Éditorial

Once again!

Early in 1992 the newsletter of the Canadian Ophthalmological Society advised its members of "The increase of scope of Optometric practice". The newsletter outlined changes in optometric legislation throughout Canada and stated, "There will be a neverending battle to defend medical and surgical treatment of eye disease from non-medical practitioners." The Society also indicated that it has obtained literature and support from their American colleagues to presumably counter Optometry's goals.

For years ophthalmology has taken an adversarial political stance with respect to Optometry and our role in vision care delivery. There have been attempts to thwart advances in our profession, including the use of anaesthetics for tonometry, the use of dilating agents for peripheral fundus evaluation (or to facilitate posterior pole evaluation) and the use of cycloplegics for refractive evaluation. Intriguingly, the use of these drugs has resulted in more effective and discriminating referrals to the very group that opposed their use. Of obvious significance, as well, is the benefit to patients of these advances in Optometry.

Once again we expect that our profession will be assailed for seeking further advances for the management of our patients. Lest I be accused of couching my terms, let me clearly state that the prescribing of therapeutic drugs by optometrists is an increase in our scope of practice that needs to and will occur. The rationalization of my belief hinges upon patient welfare, the education and training of our profession, our financially ever-flagging health care system and the increasing pressures upon ophthalmology (e.g., capping, clawbacks).

External pressures on ophthalmology have resulted in interesting changes to their practices, such as involvement with the dispensing of spectacles, the expansion of noninsured services (laser K, RK) and association with refracting physicians. Each of these changes in ophthalmology practice also warrants comment, perhaps at another time.

By now ophthalmology must understand that the diagnosis and management of disease has never been solely the purview of medicine; perhaps this is what concerns them. The position being taken by ophthalmology should not be surprising nor does it change my respect for their surgical and diagnostic abilities.

MJ Samek, OD, FAAO
Senior Editor

Encore une fois!

Au début de 1992, la Société canadienne d'ophtalmologie informait ses membres, par son bulletin, de l'«élargissement de l'étendue de l'exercice de l'optométrie». Le bulletin signalait des changements aux lois sur l'optométrie partout au Canada, et il mentionnait qu'«il y aura un lutte incessante pour défendre le traitement médical et chirurgical des maladies des yeux par des praticiens qui ne sont pas médecins». La Société a aussi indiqué qu'elle avait obtenu des documents et l'appui de ses collègues américains, vraisemblablement pour faire obstacle aux objectifs de l'optométrie.

Pendant des années, l'ophtalmologie a adopté une attitude politique d'antagonisme à l'endroit de l'optométrie et de son rôle dans la prestation des soins oculo-visuels. On a tenté de contrecarrer les progrès de notre profession, notamment l'utilisation des anesthésiques en tonométrie, l'utilisation d'agents dilatateurs pour l'évaluation périphérique du fond de l'œil (ou pour faciliter l'évaluation du pôle postérieur) et l'utilisation de cycloplégiques pour évaluer la réfraction. Fait étrange, l'utilisation de ces médicaments a donné lieu à un aiguillage plus efficace et précis auprès du même groupe qui s'y était opposé. De plus, notons l'importance manifeste de l'avantage que présentent ces progrès de l'optométrie pour les patients.

Encore une fois, nous prévoyons que notre profession sera attaquée pour avoir voulu faire progresser le traitement de nos patients. Pour ne pas qu'on m'accuse de mâcher mes mots, j'affirme haut et fort que la prescription de médicaments par les optométristes constitue un élargissement de notre pratique qui doit et va survenir. La rationalisation de mes convictions repose sur le bien-être des patients, l'éducation et la formation de notre profession, la précarité financière constante de notre réseau de soins de santé et les pressions accrues sur l'ophtalmologie (c.-à-d. le plafonnement, les récupérations).

Les pressions externes sur les ophtalmologistes ont provoqué des changements intéressants à leur pratique, comme la participation à la prescription de lunettes et l'expansion des services non assurés (laser K, RK) et l'association aux médecins spécialisés en réfraction. Tous ces changements à la pratique de l'ophtalmologie méritent aussi un commentaire; une autre fois peut-être.

L'ophtalmologie doit maintenant comprendre que le diagnostic et le traitement des maladies n'a jamais été le domaine exclusif de la médecine, c'est peut-être ce qui les inquiète. La position adoptée par les ophtalmologistes ne saurait surprendre, et elle ne modifie en rien le respect que j'accorde à leurs compétences chirurgicales et diagnostiques.

MJ Samek, OD, FAAO
Rédacteur en chef

When you've got the best Toric in the universe, word gets around.

Happy patients tell their friends...

Every day, more people are enjoying the benefits of Optima Toric lenses. Word of mouth from satisfied toric wearers can help you build your practice. And now, the simplest, most straightforward-fitting toric comes in 3 base curves, four cylinders and spherical correction from +4.00D to -9.00D, giving you the widest range of stock parameters, to fit even more patients.

If you don't have a fit set, call your Bausch & Lomb representative at 1-800-668-7510.

After all, you never know when someone new might drop in for a fitting.

Optima Toric

The toric to build your practice on.

BAUSCH & LOMB
Healthcare and Optics Worldwide

Optima Toric and Bausch & Lomb are trademarks of Bausch & Lomb Canada Incorporated.

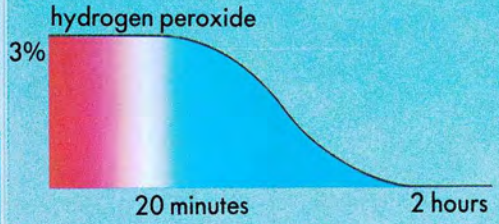
NEW ULTRACARE NOW THE BETTER 1 STEP

Simultaneous disinfection/neutralization system

1 STEP SIMPLICITY

New UltraCare 1 Step delayed release technology provides simultaneous disinfection/neutralization. Patients will never have to worry about when to change the disc again.

1 STEP COMFORT



New UltraCare 1 Step tablets supply fresh, full strength neutralization power every time, no stinging or burning from residual peroxide left by an ineffective or worn out disc. *AND* UltraCare 1 Step is Preservative Free.

RECOMMEND NEW

ULTRACARE™

NOW THE BETTER 1 STEP

ALLERGAN OPTICAL

President's Podium

Mot du président

Optometry's celebration of 25 years at Waterloo—a time to reflect and to plan for the future

When it was officially announced at CAO's 1967 Biennial Congress in Montreal that the College of Optometry of Ontario would be moving from St George Street in Toronto to the University of Waterloo, it was a euphoric occasion. Immediately, this historic event triggered a great deal of optimism for the continued growth of the profession in Canada, optimism that would prove to be well founded.

The quarter century that has since passed has been, indeed, an exciting time for both the School and Optometry itself.

We have seen the School grow and develop in order to meet the new challenges of the changing profession. It is now acknowledged around the world for its many achievements and is seen as a leader in many of the evolutionary and groundbreaking changes that have since advanced Canadian Optometry's contribution to global health care.

Healthy, too, is the relationship that has developed between the School and Canada's national and provincial associations of optometrists. It is almost a partnership.

We in practice know we can depend on the faculty for support and expert advice. Its resources have always been readily available to the profession. In return, Canadian Optometry has given the School its full support. Granted, occasionally the Association has supplied some advice not always solicited, and so perhaps we might more accurately be said to share a family relationship, rather than a partnership.

Optometrists responded enthusiastically to the challenge of raising funds for the new building, and the University continues to recognize its 25 years of optometric alumni as valuable assets. The alumni, in turn, have always been extremely proud of our School and continue to support it.

More than 15 years ago, the recognition of the need for organized, national support for optometric education and research led to the establishment of the Canadian Optometric Education Trust Fund (COETF). The School has since benefited from annual COETF awards, and Canadian Optometry has benefited even more. The COETF has been able to give support to students in graduate studies, and to supply funds for research projects at all levels of the program.

L'optométrie célèbre ses 25 ans à Waterloo — un moment pour réfléchir et planifier l'avenir

Au Congrès biennal de l'ACO de 1967 à Montréal, lorsqu'on a annoncé officiellement que le College of Optometry of Ontario déménageait de la rue St George de Toronto à l'Université de Waterloo, ce fut l'euphorie. Immédiatement, cet événement historique a entraîné une vague d'optimisme par rapport à la croissance constante de la profession au Canada, un optimisme qui allait se révéler bien fondé.

Le quart de siècle qui s'est écoulé depuis a été une période particulièrement stimulante pour l'école et l'optométrie elle-même.

Nous avons vu l'école croître et se développer afin de répondre aux nouveaux besoins d'une profession en changement. L'école est maintenant reconnue à travers le monde pour ses nombreuses réalisations, et on la considère comme un chef de file par rapport à un grand nombre de changements évolutifs et innovateurs qui ont depuis élevé la contribution de l'optométrie canadienne au rang de soin de santé global.

De plus, une «saine» relation s'est établie entre l'école et les associations d'optométristes nationale et provinciales du Canada. C'est un quasi-partenariat.

En pratique, nous savons qu'on peut se fier aux professeurs pour obtenir un appui et des conseils éclairés. Les ressources de l'école sont toujours facilement accessibles à la profession. En retour, l'optométrie canadienne a donné son plein appui à l'école. Nous reconnaissons que l'Association a parfois donné des conseils sans qu'on le lui demande, et il serait peut-être ainsi plus juste de dire que nous partageons une relation familiale plutôt qu'un partenariat.

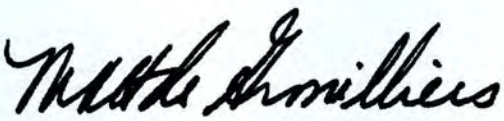
Les optométristes ont réagi avec enthousiasme à l'invitation de réunir des fonds pour le nouvel immeuble, et l'université continue de reconnaître ses 25 années d'anciens de l'optométrie comme des atouts précieux. En retour, les anciens ont toujours été extrêmement fiers de notre école et ils continuent de l'appuyer.

Il y a plus de 15 ans, la reconnaissance de la nécessité d'un appui organisé et national pour la formation et la recherche en optométrie a donné lieu à la création du Fonds de fiducie des optométristes canadiens pour l'éducation (FFOCE). Depuis lors, l'école a bénéficié de bourses annuelles du FFOCE, et l'optométrie canadienne en a profité plus encore. Le FFOCE a été en mesure d'aider les étudiants des deuxième et troisième cycles et

Now, with Optometry having reached this 25-year milestone in our development, it is my observation that we are anticipating the next 25 years will be just as exciting as those that have transpired since I heard that 1967 announcement.

Optometry, if and when it is confronted with potential setbacks, has a history of converting them to challenges and opportunities. We are not made pessimistic nor complacent by them; indeed we are stimulated and, inevitably, we emerge a little more knowledgeable and a lot more experienced. In 2017, when we look back on 50 years of Optometry at Waterloo, I know we will be every bit as pleased then with what has been achieved as we are today.

I congratulate the staff, faculty and students at today's School of Optometry, University of Waterloo, and, along with your many, many colleagues and classmates, wish you nothing but the best in the next quarter century!



Dr Margaret Hansen des Groseilliers
President

de financer des projets de recherche à tous les niveaux du programme.

L'optométrie ayant maintenant franchi cette étape de 25 ans d'évolution, permettez-moi de déclarer que nous prévoyons que les 25 prochaines années seront tout aussi stimulantes que la période écoulée depuis que j'ai entendu ce communiqué en 1967.

Lorsqu'elle fait face à des échecs éventuels, l'optométrie les a toujours transformés en défis et en occasions. Ils ne nous rendent pas pessimistes ou suffisants; en effet, nous sommes stimulés et, inévitablement, nous en ressortons un peu plus sages et beaucoup plus expérimentés. En 2017, lorsque nous ferons une rétrospective des 50 ans d'optométrie à Waterloo, je sais que nous serons alors tout aussi heureux de nos réalisations que nous le sommes aujourd'hui.

Je félicite le personnel, les professeurs et les étudiants qui œuvrent actuellement à l'école d'optométrie de l'Université de Waterloo, de même que vos très nombreux collègues et condisciples. Je vous souhaite la meilleure des chances au cours du prochain quart de siècle!

Dr Margaret Hansen des Groseilliers
Présidente

Clinical Diagnosis Diagnostic clinique

Catherine Chiarelli, OD, FAAO
Vision Institute

DR, a 33-year-old white female, presented with complaints of poor night vision and occasional vertical diplopia while reading. These symptoms had become worse over the past year. Spectacles had been prescribed each year since childhood, but were worn inconsistently. She felt that her current prescription was not helpful and did not use it.

Ocular health history was negative for trauma and disease. She denied any family history of ocular disease or vision loss. General health history was positive for long-standing headaches, which were associated with numbness on the right side of the face and muscle tension in the neck. She also had asthma.

Unaided visual acuity:	OD 20/200 OS 20/50+
Refraction:	OD PLANO -3.00 X 166 $20/25^{-3}$ OS -1.00 -0.75 X 060 $20/20^{+3}$
Binocularity:	non-strabismic; orthophoria
Anterior Segment:	within normal limits
Pupil Reflexes:	normal; no afferent defect
Fundus:	see photos

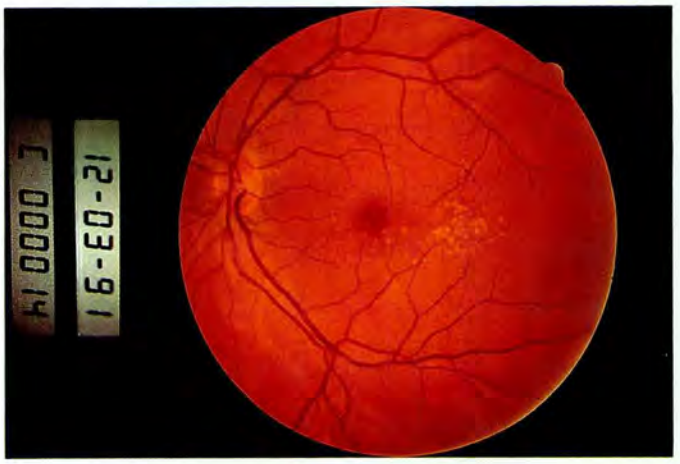


Figure 1. Left eye.

ERG:	normal photopic and scotopic responses
Fluorescein angiography:	staining of drusen, without leakage of sub-retinal fluid
Amsler grid:	normal, no distortion noted

What is the diagnosis?
What is the prognosis?
What management is appropriate?
(See page 199.)

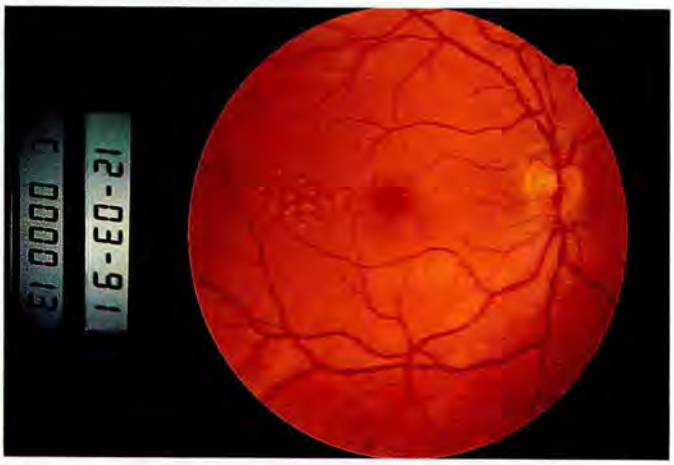


Figure 2. Right eye.

Vision Awareness Week (March 1–7) 1993

As this year's poster indicates, we're hoping to have some fun—but with a serious topic. Vision Safety is important on the job and at home. This year's poster includes images that stress the importance of thinking about eye protection—working, playing or just "catching some rays".

This year's Honorary Chairman is **J Arthur St-Aubin, President of the Canadian Centre for Occupational Health and Safety (CCOHS)** in Hamilton, Ontario. (That's him on the pamphlet.) The CCOHS is a vital Canadian resource for a wide range of job-related injury, safety and accident prevention information. Their linking with our Vision Awareness Week campaign in 1993 will provide valuable support for both our organizations.

By this time all members should have received their poster and pamphlet for Vision Awareness Week 1993. For extra copies of the poster and pamphlet, please contact Chantal Wall at CAO. Prices are as follows:

1. If you order the same number of posters and pamphlets, your price is $1.40 per unit (a "unit" being one poster plus one pamphlet).

2. If you order posters only, your price is $1 per poster. If you order pamphlets only, your price is $.50 per pamphlet. (**Note:** If you order, for example, 50 posters and 100 pamphlets, you will be invoiced for 50 units plus 50 extra pamphlets.)

Shipping, GST and Ontario PST (8%), if applicable, is **EXTRA** and will be added to the invoice when your order is filled.

Semaine de la vision (du 1er au 7 mars) 1993

Nous espérons avoir du plaisir comme le montre l'affiche de cette année — mais nous avons aussi un sujet sérieux. La sécurité de la vision est importante au travail et à la maison. Cette année, les images de l'affiche montrent l'importance de penser à la protection de l'oeil — en travaillant, en jouant ou simplement en «prenant du soleil».

Cette année, le président honoraire est **J. Arthur St-Aubin, président du Centre canadien d'hygiène et de sécurité au travail (CCHST)** d'Hamilton (Ontario). (On le voit sur la brochure.) Le CCHST est une source d'information canadienne indispensable sur la prévention des accidents, sur la sécurité et sur un large éventail de blessures liées au travail. Il a noué un lien avec notre campagne de la Semaine de la vision 1993 qui fournira un soutien précieux à nos organismes.

Tous les membres devraient maintenant avoir reçu leur affiche et leur dépliant. Pour obtenir des exemplaires supplémentaires, veuillez communiquer avec Chantal Wall à l'ACO. Les prix sont les suivants :

1. Si vous commandez le même nombre d'affiches et de brochures, le prix est de 1,40 $ l'unité (c.-à-d. une affiche et une brochure).

2. Si vous commandez des affiches seulement, le prix est de 1 $ l'affiche. Si vous commandez des brochures seulement, le prix est de 0,50 $ la brochure. (**Note :** si vous commandez 50 affiches et 100 brochures par exemple, vous recevrez une facture pour 50 «unités» et 50 brochures supplémentaires.)

Frais d'expédition, TPS et TVP de l'Ontario (8 %) s'il y a lieu, **EN SUS**, ajoutés à la facture lorsque votre commande est prête à livrer.

QUALITY

Some Take It Too Lightly.

LUXOTTICA® QUALITY

WE'VE MADE IT THE FOUNDATION OF OUR STRENGTH.

We recognize that quality remains a fundamental
component of frame wearer satisfaction.
And satisfied customers come back.
That is why the Luxottica Group is committed
to continually enhancing — never compromising —
the quality of its frames.

GROUPE
LUXOTTICA®
GROUP

Corneal dystrophies and degenerations Part 2: The stroma

Monika Braun, William M Lyle

key words: stromal dystrophies; clinical signs; histological features; inheritance pattern

mots clés : dystrophies du stroma; signes cliniques; caractéristiques histologiques; profil héréditaire

Part 2 in our continuing review of corneal dystrophies deals with stromal dystrophies. An earlier paper (Part 1) discussed dystrophies of the anterior layers of the cornea and a subsequent paper (Part 3) will do the same for dystrophies involving the posterior layers. A table simplifies comparisons among the 16 or more different stromal dystrophies and related degenerations.

La partie 2 de notre examen des dystrophies cornéennes porte sur les dystrophies du stroma. Un premier article (partie 1) a abordé les dystrophies des couches antérieures de la cornée, et un article subséquent (partie 3) fera de même pour les dystrophies des couches postérieures. Un tableau simplifie les comparaisons entre au moins 16 dystrophies distinctes du stroma et les dégénérescences connexes.

M Braun, BSc, Optometry student
WM Lyle, OD, PhD, FAAO — School of Optometry, University of Waterloo

The stroma constitutes the largest portion of the corneal tissue (90% of its thickness). The stroma can be affected adversely by various systemic conditions as well as by disorders of the anterior and posterior corneal layers. Dystrophies and degenerations involving other layers of the cornea often extend into or otherwise affect the stroma. For example, the dermo-chondro-corneal dystrophy of François (AR), which affects all corneal layers, has its main effect in the anterior stroma.

In lecithin cholesterol acyltransferase (LCAT) deficiency (AR), also called Norum disease, grey lipid dots are visible in both axial and peripheral stroma.

In most types of ichthyosis, corneal opacities appear. In the AD type the opacities are in the stroma. In the XR type one sees grey opacities in the deep stroma in patients with the disease; similar opacities are present in female carriers of this gene.

The vacuoles in the epithelium and stroma of patients with the Hunter type of mucopolysaccharidosis (XR) are followed by corneal deposits. Carriers may have whorl-like corneal opacities.

Keratan sulfate accumulates in the stroma in Morquio syndrome (AR).

As in Part 1, we use the following abbreviations: AD for autosomal dominant, AR for autosomal recessive, XR for X-linked recessive, XD for X-linked dominant and S for those conditions that appear to arise sporadically. For a condition that is inherited by means of genes in the mitochondria, the designation Mito is used.

Central cloudy dystrophy

Central cloudy stromal dystrophy is rare, bilateral, symmetrical, AD or S, and nonprogressive. First described in 1956 by François, this dystrophy may be observed in affected individuals from 8 to 87 years of age.[1] Grey, snowflake-like opacities are visible in the axial region of the stroma, but visual acuity is not reduced.[2]

The opacities form multiple, cloudy, grey areas with indistinct margins and an indefinite structure. The clouds in the axial stroma are separated by transparent crack-like zones, while the corneal periphery remains clear.[1,2] Although the lesions are most dense in the deep posterior stroma, they may involve the entire stroma. The poorly defined posterior stromal opacity becomes less dense as it extends anteriorly.[3] Sclerotic scatter helps to make these opacities visible.

No histopathology reports or adequate explanations of the pathogenesis of central cloudy dystrophy are available.[1,2] It is unaccompanied by pain or inflammation. Corneal sensitivity and visual acuity remain normal. No treatment is required for this dystrophy.[1]

In 1961, Waardenburg and Jonkers described a progressive, AD dystrophy in which snowflake or hailstone-like opacities first appeared in the axial stroma

but later spread to the Bowman and epithelial layers. See honeycomb dystrophy in Part 1.

Central cloudy dystrophy resembles posterior crocodile shagreen; however, the central cloudy opacity is poorly defined posteriorly and it extends anteriorly, while posterior crocodile shagreen forms a discrete posterior stromal layer.[3] The designation central can be confusing. Does it mean the axial, pupillary, interpalpebral region or does it mean central in terms of fore and aft position in the corneal stroma?

Central crystalline dystrophy

Central crystalline or Schnyder dystrophy (also spelt Snyder, Snijder and Schnijder) is an axial, AD or S, stromal dystrophy. The gene may be on chromosome 9. Subtle, asymptomatic, crystalline deposits appear in the anterior axial stroma in the first year of life but usually remain undetected until the third or fourth decade. In these later decades diffuse, axial haziness reduces the visual acuity; however, neither inflammation nor irritation is present.[1,4,5] This dystrophy is frequently accompanied by a prominent bilateral arcus lipoides. Corneal sensitivity is slightly diminished but erosions do not occur. Genu valgum accompanies the corneal dystrophy as an independently inherited trait in some families. Central crystalline dystrophy may not be a component of a systemic disease, but rather a local disorder of corneal lipid metabolism. Although hyperlipidemia and xanthelasma have been reported in some families, others found no association between these conditions and central crystalline dystrophy. About 50% have elevated serum lipids.[6] The cause of central crystalline dystrophy is unclear. Possibly three subtypes exist: limited to the cornea, with hyperlipidemia and with genu valgum. Weller and Rodger[5] believe that defective clearance of cholesterol from the cornea is responsible. Brownstein et al.[6] suggest overproduction of lipid by keratocytes or epithelium.

Crystalline dystrophy has several corneal components: minute, diffuse, grey, crystals forming an axial, disc-shaped or ring-shaped stromal haze, often accompanied by a dense corneal arcus (80%) and white limbal girdle of Vogt (80%). The crystalline deposits are fine, needle-like crystals that glisten yellow, blue, green and red. The lipid is intracellular and extracellular. The deposits aggregate just below the Bowman layer, sometimes in a wreath-like formation in the axial portion of the cornea, and may extend deep into the stroma. The peripheral stroma is usually clear; however, in some of these eyes the stroma contains random, small, white, punctate opacities.[1] The opacification stops progressing after the third or fourth decade, and the number of crystals may decrease.

Histologically, the epithelium is normal. The basement membrane is sometimes intact; however, in most cases the basement membrane has areas of focal thickening and other areas where the membrane is absent. The Bowman layer is disrupted by numerous, cholesterol-containing, oblong holes with rounded corners. The Bowman layer may be very thin or absent and tends to become scarred. The anterior stroma has larger, more compact spaces, which are similar in shape to those in the Bowman layer. These holes are filled with densely packed, polychromatic, needle-like, unesterfied cholesterol crystals. The entire stroma also contains smaller, round, empty spaces which represent dissolved lipid deposits.[1,4–7] Scarring also occurs in the stroma. The keratocytes are abnormal. Some have membrane-bound oval structures in their cytoplasm. Numerous ovoid vesicular formations form an almost continuous layer between the deep posterior stroma and the Descemet membrane. The Descemet membrane and the endothelium are generally said to be unaffected by central crystalline dystrophy.[1,4,5] However, Freddo et al.[8] studied the posterior layers of a cornea affected by this dystrophy and found that the deeper layers were affected. Rare, focal areas of degenerated endothelial cells constituted small discontinuities in the endothelial cell covering of the Descemet membrane.

In central crystalline dystrophy, keratoplasty is rarely required, as the reduction in visual acuity is not severe.[1] The lipid opacities can appear in the grafted tissue.[6] Compare with Bietti marginal crystalline dystrophy.

Hypergammaglobulinemia can cause crystalline corneal deposits and so can childhood cystinosis, hyperuricemia, oxalosis, tyrosinosis, porphyria and calcium deposition. In Tangier disease (AR), a disorder of lipid metabolism, white, dot-like opacities are found in the deep stroma more in the axial region than in the corneal periphery.[9] Somewhat similar white dots appear in the stroma in lecithin-cholesterol acetylase deficiency and in another disorder of lipid metabolism called fish eye disease.[9]

Congenital hereditary stromal dystrophy

Congenital hereditary stromal dystrophy (CHSD) is a bilateral, or rarely unilateral, symmetrical, AD or S, nonprogressive corneal dystrophy that, as its name indicates, is present at birth. Probably fewer than 10% of corneal dystrophies are of this type. The superficial, feathery or flaky, diffuse, cloudy opacity, is densest in the anterior axial stroma, and fades toward the periphery.[1,4,10] Searching nystagmus (often accompanied by esotropia) results from the early decrease in visual acuity.[1] The cause of congenital hereditary stromal dystrophy is unknown.[1] Witschell et al.[10] postulate that the feathery clouding is the result of the abnormal distribution and composition of stromal glycosaminoglycan. Others suspect a disorder of stromal fibrogenesis.

Histologically, the stroma contains two types of abnormal collagen lamellae with fibrils of unusually small diameter (15 nm) as compared to the normal size of 30 nm.[1,4,10] Tightly packed lamellae with highly aligned

collagen fibres alternate with loose, haphazardly arranged collagen fibrils enmeshed in an amorphous, lucent ground substance. Corneal thickness is normal. The Descemet membrane is normal except for the poorly developed anterior banded portion. The smooth regular epithelium, the Bowman layer and the endothelium are all normal.[1,4,10]

CHSD is treated by penetrating keratoplasty. Generally the visual acuity improves only to 6/60, however, occasionally acuities as good as 6/9 have been reported.[1]

A congenital, bilateral, AR, stromal dystrophy has been confused with congenital glaucoma.[11] Although the axial portion of the stroma is somewhat edematous, vision may be as good as 6/21. See also congenital hereditary endothelial dystrophy and Fuchs endothelial-epithelial dystrophy.

Cornea farinata

Cornea farinata of Vogt (1930) is a scattering of discrete, tiny, grey to white, flour-like opacities throughout the deep corneal stroma.[12–14] The opacities are axial, bilateral and asymptomatic. They are seen best by retroillumination. The condition is AD and does not decrease corneal sensitivity or visual acuity.[12,14] The cause of cornea farinata is unknown; however, it is thought to result from aging.[12,14] Cornea farinata is probably a degenerative disease.

The posterior stromal keratocytes contain lipofuscin-like material which results from aging and the natural "wear and tear" of the cells.[12,13] Cornea farinata opacities are quite similar to those seen in pre-Descemet membrane dystrophy and some have classified the floury opacities as part of pre-Descemet dystrophy.[15]

A somewhat similar deep punctate dystrophy was described by Franceschetti and Maeder in 1954 and by François and Schlaeppi in 1957. Both patients had ichthyosis.[16]

Dermo-chondro-corneal dystrophy

Dermo-chondro-corneal dystrophy (AR), as described by François[17] in 1949, results in minute, axial, whitish or slightly brownish, punctate opacities in all corneal layers but especially the anterior stroma.[18] Visual acuity is reduced. The systemic effects are osteochondral dystrophy of the extremities and cutaneous xanthomas on the back of the hands and fingers, on the dorsal aspect of the elbows and on the nose and ears. The osseous changes are detectable in the first decade. A few patients have nephropathy and proteinuria. An abnormal EEG and seizures are also reported.

Fleck dystrophy

Fleck, speckled, mottled or mouchetée corneal dystrophy was described in 1956 by François and Neetens.[4,19,20] This rare stromal dystrophy is AD and bilateral; however, very asymmetrical or even unilateral cases have occurred.[1] The gene shows high penetrance and variable expressivity. Fleck dystrophy can be detected at two years of age and may be congenital. The cause of fleck dystrophy is unknown. The epithelium is smooth and the opacities are small; therefore, the finding is usually incidental or may be missed unless a careful slit lamp examination is done. Both visual acuity and corneal sensitivity are normal.[1,20–22] The opacities remain constant in each individual as the condition is not progressive.[1,4]

In optical section, the flecks appear as discrete, flat, grey-white snowflakes or dandruff-like particles scattered out as far as the limbus in all stromal layers. Among those affected, the opacities vary in size, shape and number, and each is surrounded by clear, unaffected stroma. Most lesions are stellate, comma, oval or circular in shape with sharp, discrete margins, while others have edges that are scalloped. Each individual fleck is a discrete white ring surrounding a clear centre, like a wreath.[1,21,22] These opacities are seen best against the red reflex from the fundus. Opacities are more numerous axially and in midstromal and deeper layers.

Histologically, the stroma is normal except for certain swollen keratocytes containing two abnormal substances. Excess glycosaminoglycan is found in membrane-bound intracytoplasmic vacuoles containing fibrogranular material. Lipids are also present in smaller membrane-bound vacuoles containing electron-dense lamellar bodies. The abnormal keratocytes scattered throughout the entire stroma correspond to the grey-white flecks. The epithelium, Bowman layer, Descemet membrane and endothelium are all normal.[1,4,21]

Since this benign stromal dystrophy is asymptomatic and does not affect visual acuity, no treatment is required.[1] Similar flecks have been reported with central cloudy corneal dystrophy, keratoconus, limbal dermoids and pseudoxanthoma elasticum. See also the flecks associated with epithelial basement membrane dystrophy and the lesions of deep filiform dystrophy.

Granular dystrophy

Granular (Groenouw nodular Type 1 or Bücklers Type 1 or Fleisher) corneal dystrophy is bilateral, AD or S, and symmetrical. Clinically granular dystrophy shows interfamilial variability. Granular dystrophy constitutes about 15% of all cases of corneal dystrophy. The cause of granular dystrophy is still under discussion. An involved gene has been mapped to chromosome 12p. In the first decade of life protein or phospholipids accumulate in the axial region of the anterior corneal stroma.[1,4,23,24]

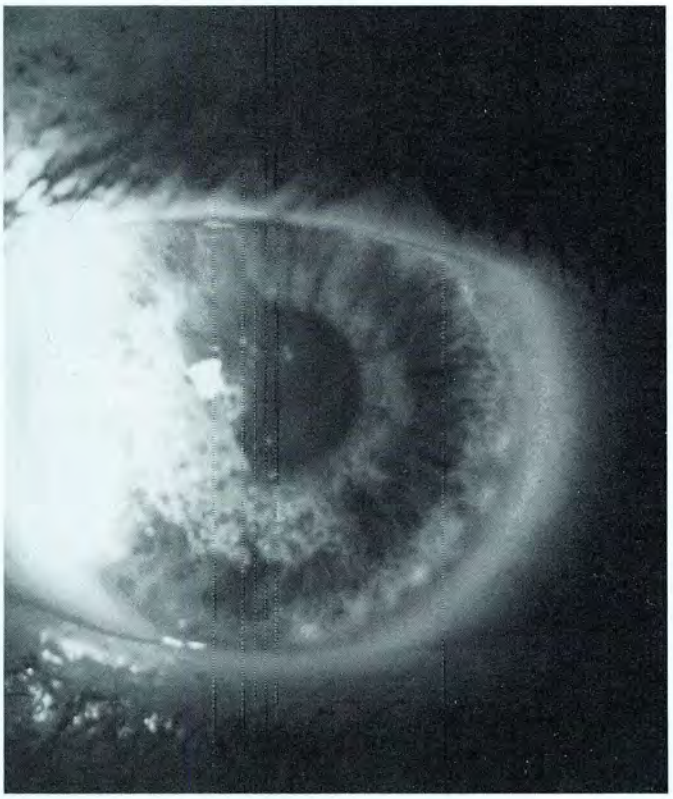


Figure 1. Granular dystrophy

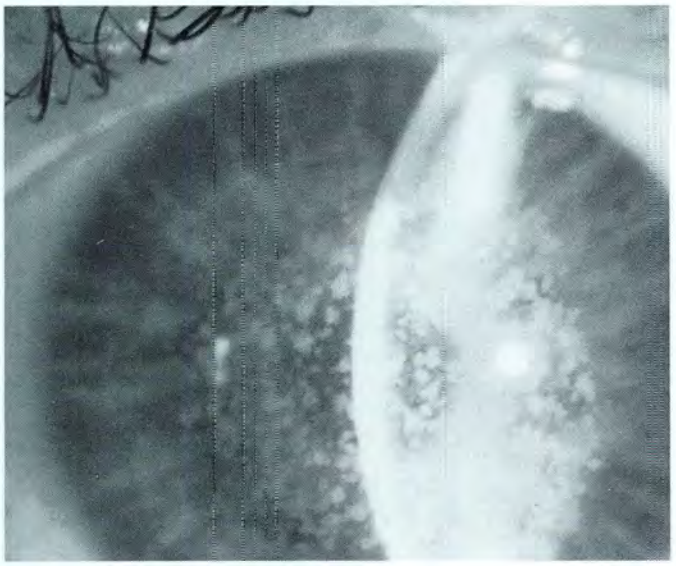


Figure 2. Granular dystrophy

Beneath the Bowman layer, discrete, focal, round or oval, grey-white spots, 0.2 to 0.4 mm in diameter,[1] appear. In a young patient, the stroma between the white, granular opacities is clear and the epithelium is regular; therefore, the patient is asymptomatic and has normal visual acuity. The opacities are predominantly in the anterior layers of the axial stroma, with a few in the posterior stroma (especially in older patients) and none in the periphery.[25] The spots are usually scattered

Figure 3. Granular dystrophy

randomly but sometimes form linear rows or circular chains especially in the young. The white spots appear as solid, nontranslucent or powdery, semitranslucent or ring-shaped opacities with centres containing tiny, grey-white fragments (figure 1). As the patient ages, the initial opacities enlarge and become more dense, and additional opacities develop deeper in the stroma (figure 2). The stroma between the grey-white, bread-crumb-like spots opacifies, causing the visual acuity to decrease gradually to 6/60.[1,23,26] In the fifth decade, the anterior stroma of an affected cornea presents a diffuse, irregular, ground-glass, hazy appearance, sometimes described as snowflakes in a cloud in the axial region (figure 3).[1] The Bowman layer is damaged in some places. The epithelium becomes irregular and corneal sensitivity is reduced. Some of these eyes suffer epithelial erosions.[24,26]

Histologically, the corneal epithelium and basement membrane may vary in thickness, although they are usually smooth.[1,23] Focal aggregates of eosinophilic, granular, hyaline deposits, that stain brilliant red with the Masson trichrome stain, are found at all levels of the corneal stroma and some in the Bowman layer.[1,4,23,24,26] These extracellular, subepithelial, electron-dense, rod-shaped, 100- to 500-µm wide, crystalline deposits are round or oval in cross-section and are surrounded by 8- to 10-nm microfibrils. Later, the electron-dense deposits

infiltrate between the epithelial cells, resulting in a homogeneous appearance. Histochemically, the electron-dense deposits are abnormal, noncollagenous proteins containing tyrosine, tryptophan and sulfur-containing amino acids.[1,23] The keratocytes containing the electron-dense material undergo degenerative changes.[1,4,23] Other deposits between the stromal lamellae or within the cytoplasm of keratocytes give the cornea a homogeneous or moth-eaten appearance. Corneas with granular dystrophy have an increased lipid content of every phospholipid class and have antibodies to microfibrillar protein.[4,23] Rodriques and Krachmer[4] speculate that the deposits of phospholipids result from a genetic defect in the processing or assembly of membrane-derived corneal epithelial phospholipids and proteins. The serum level of keratan sulfate is very low. Wittebol-Post et al.[23] support the view that the keratocyte is the primary source of the abnormal electron-dense material. The Descemet membrane and the endothelium appear to be normal.

Waardenburg and Jonkers in 1961 described a progressive variant of granular corneal dystrophy.[27] In this AD dystrophy, the minute, snowflake-like spots appear first in the stroma and later in the Bowman layer and epithelium. There are no clear areas between the opacities. The affected child has episodes of irritated, red, photophobic eyes but the attacks become less frequent as the patient ages even though the changes in the epithelium, Bowman layer and axial stroma worsen. Another variant affects the superficial corneal layers and resembles ring-shaped corneal dystrophy. Granular dystrophy may resemble Reis-Bücklers dystrophy.

Clinical severity of granular dystrophy is variable. Keratoplasty is usually necessary for patients (in their fifth or sixth decade) suffering from granular dystrophy and it has a favourable prognosis. Occasionally patients develop recurrences 1 to 19 years postoperatively.[1,25]

Granular corneal dystrophy can occur with anhidrotic or hypohidrotic ectodermal dysplasia (AR, AD, XR), and with Christ-Siemens-Touraine syndrome (S, AD, AR).

Figure 4. Keratoconus

Figure 5. Keratoconus, Munson sign

Keratoconus (conical cornea)

Keratoconus, a bilateral disorder affecting all layers of the cornea, is characterized by progressive apical thinning and central ectasia which result in a cone-like anterior protrusion and irregular myopic astigmatism.[28–30] Keratoconus may be acquired (S), but 10 to 15% of cases show a familial tendency.[28] The probable form of transmission is AD; however, AR is sometimes indicated. It affects about 1/1,000 in the population. Keratoconus is more likely to develop in females. Keratoconus appears in the second decade,[30] asymptomatically or with vision impairment. This slowly progressive dystrophy produces either obvious or minor morphological changes in the cornea.[28] Typically, the progression of keratoconus continues for only 5 or 6 years. In most cases of keratoconus the cornea assumes one of two shapes.[28,31] The more common form is a round ectatic area with a limited diameter and a large degree of conicity; the cone centre is located in the inferior nasal quadrant (figures 4 and 5). The other corneal cone type is oval. It is larger and its apex is closer to the corneal periphery in the inferotemporal quadrant. In advanced keratoconus, marked thinning of the corneal stroma is apparent as one approaches the cone apex.[28,29] The stroma is one-third or less its normal (0.5 mm) thickness at the apex of the cone. Ruptures in the Bowman layer cause permanent linear scars at the apex of the cone which reduce the vision. Hydrops develops as a result of acute ruptures of the Descemet membrane and produces stromal and

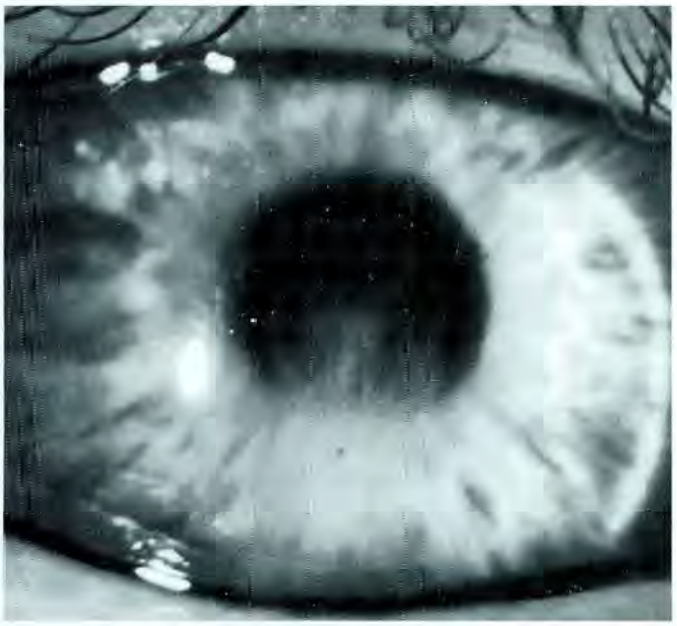


Figure 6. Keratoconus, Vogt striae

epithelial edema.[28,30] This may lead to scar formation. The most common scarring pattern is a fine, reticular network in the Bowman layer and stroma which has little effect on vision. A brown Fleischer ring, caused by iron deposition in the epithelium, may develop at the base of the cone.[31] There is increased visibility of the stromal nerves and Vogt vertical striae (figure 6).[28,29] These wider greyish lines are situated in the posterior third of the stroma and may be stretch lines.

The cause of keratoconus is difficult to determine because of the problem of distinguishing between the primary event initiating keratoconus and the subsequent changes.[28] Some studies have shown increased protein biosynthesis, but Lawless et al.[28] suggest that despite the increased synthetic activity, the slow destruction and thinning is caused by increased collagenolytic activity. The presence of amyloid has been reported.[32]

Histologically, the basal epithelial layer is edematous and pale. The basal cells eventually disappear and leave behind one or two layers of superficial epithelium. The basement membrane is fragmented in many areas. the Bowman layer is composed of very fine multiple fibrils. Early in the disease, swelling, fibrillar degeneration and breaks occur in the Bowman layer and these fill with the underlying stromal collagen. There are lesions throughout the Bowman layer and the stroma.[28,33] The superficial stroma is replaced by irregularly arranged connective tissue. Until the Descemet membrane ruptures late in the disease, it shows no histological changes. However, soon afterward the ruptured edges curl anteriorly toward the stroma.[28,33] Folds or ruptures in the Descemet membrane are called Haab striae and tend to be horizontal. Later the ruptured areas are covered by endothelial cells from adjacent sites and new Descemet membrane is laid down.[29] The endothelial cells are flat and their nuclei are far apart.

Keratoconus occurs with increased frequency in patients with aniridia, blue sclerae, congenital cataract, corneal degenerations, ectopia lentis, microcornea,[28] pre-Descemet membrane dystrophy and endothelial dystrophy. A relation to allergy and to eye rubbing has been suggested. Compare with pellucid marginal degeneration. Keratoconus can be associated with conditions such as Down syndrome and with various systemic connective tissue disorders, including Apert syndrome (S,AD,AR), atopic dermatitis, Besnier purigo (S), Crouzon syndrome (AD,S), Ehlers-Danlos syndrome (type VI,AR), Leber optic atrophy (Mito), Marfan syndrome (AD,S), osteogenesis imperfecta (AD,AR,S)[28,34] and pseudoxanthoma elasticum.

Iron deposits accumulate in the cornea in several other conditions including Coats white ring, Ferry line, Hudson-Stahli line and Stocker line, and after an iron-containing foreign particle has entered the cornea.

Patients with mild forms of keratoconus can be helped with spectacles. However, advanced keratoconus requires rigid contact lenses and, if the disease progresses, penetrating keratoplasty.[28,30] The use of epikeratoplasty is advocated for patients who do not have advanced keratoconus and are contact lens intolerant.[30] Risk to first degree relatives of a patient with keratoconus is about 10%. Compare with keratoglobus, in which the entire cornea becomes thinner.

Lattice corneal dystrophy

Three or four different varieties of inherited lattice or reticular corneal dystrophy (LCD) have been described: LCD type I (Biber-Haab-Dimmer), LCD type II (Meretoja) and LCD type III.[35–37] Type I is bilateral, slowly progressive and AD or rarely AR. The local accumulation of amyloid is a primary amyloidosis, unrelated to a systemic disease. Type I lattice dystrophy (once called Bücklers type III) is recognizable in the first decade of life when only superficial, punctate, stromal opacities are present. It is characterized by three distinct slit-lamp observations: superficial spots, a diffuse, axial, stromal haze, and tiny refractile lines and spots.[4,35] Histologically, in type I the epithelium and basement membrane are abnormally thinned as they are in most conditions involving recurrent erosions. The Bowman layer is thickened and partially replaced by amyloid deposits and irregular connective tissue. The deposits contain a protein of a type designated AP. The AP protein is a structural protein, related to the immunoglobulins and present in primary and secondary amyloidosis. The amyloid deposits are concentrated anteriorly with a few deeper in the stroma. The keratocytes frequently undergo degeneration. Ultrastructurally, the amyloid deposits are electron-dense, extracellular filaments ranging from 8 to 10 nm in diameter.[4] Painful

Table 1. Stromal dystrophies and degenerations

Descriptive term	Common eponym	Layer(s)	Vision impaired	Age of onset	Mode of inheritance	Bilateral/ Unilateral	Description of the opacity	Abnormal substance(s)
Central cloudy dystrophy	François	Posterior layers of axial stroma but extends anteriorly	No	Any	AD,S	B	Small grey, snowflake-like spots with polygonal shape	None identified
Central crystalline dystrophy	Schnyder	Bowman layer, and anterior axial stoma. Periphery clear, but often have a corneal arcus	Yes, eventually but mildly	1st decade	AD,S	B	Minute, crystalline, needle-like, discoid or ring-like opacities. Increase to form stromal haze	Cholesterol, lipid deposits
Congenital hereditary stromal dystrophy (CHSD)	—	Stroma, and anterior banded layer of Descemet membrane	Yes	Congenital	AD,S	B/U (rare)	Feathery, flaky, diffuse opacity in axial stroma but can affect all the stroma	Glycosaminoglycan, collagen filaments are abnormal
Cornea farinata	Vogt	Deep stroma. (See pre-Descemet dystrophy.)	No	Later in life	AD	B	Flour-like, tiny, grey-white, axial opacities	Lipofuscin-like. Degenerative
Dermo-chondro-corneal dystrophy	François	All layers but especially the anterior stroma	Yes	1st decade	AR	B	Whitish or slightly brownish punctate opacities in all corneal layers	Lipid?
Fleck, speckled or mouchetée dystrophy	François & Neetens	All stromal layers, axial and peripheral regions. Often said to be nonprogressive.	No	1st decade, may be congenital	AD	B/U (rare)	Minute, grey-white, ovoid or circular opacities may increase in number	Glycosaminoglycan, lipids in keratocytes
Granular or nodular dystrophy	Groenouw Type I, Bücklers Type I, Fleisher Type	Anterior, axial stroma; clear periphery; a few opacities in posterior stroma. Bowman and epithelial layers affected later. Epithelial erosions.	Yes	1st decade	AD,S	B	Large, discrete, focal, white, crumb-like opacities. Later stroma between nodules becomes cloudy, the initial opacities become larger, "snowflakes in a cloud".	Phospholipids, protein, hyaline in sub-epithelial extracellular pockets. Deposits also in keratocytes
Keratoconus	—	All layers of the cornea. Ruptures in Bowman and Descemet layers, Haab straie	Yes	2nd decade	S,AD,AR	B	Cone-like anterior protrusion with stromal thinning, edema and scarring	Changes in protein and collagen. Some degeneration
Lattice or reticular corneal dystrophy	Type I Biber-Haab-Dimmer	Bowman layer, anterior stroma, clear periphery; epithelial erosions, keratocyte degeneration	Yes	1st decade	AD, (AR rare)	B	Thin, branching, streak-like opacities. Axial stromal haze. Rod-like, grey lines which thicken; scarring	Amyloid in anterior stroma and beneath epithelium
	Type II Meretoja or Finland	Bowman layer, anterior stroma; epithelial erosions	Usually not until late in life	3rd decade	AD	B	Milder than in Type I but radial lines longer than in Type I and extend to periphery	Amyloid in cornea, conjunctiva and eyelids
	Type III	Bowman layer, stroma. Leaving a narrow rim clear at the periphery	Yes	7th to 9th decade	AR	B	Very thick, radial lines. Slow progression. Large stromal deposits	Amyloid in mid-stroma
	Type IIIA	Similar to Type III but have corneal erosions	Yes	7th to 9th decade	AD	B	Similar to Type III	Amyloid

Table 1. Stromal dystrophies and degenerations (cont'd)

Descriptive term	Common eponym	Layer(s)	Vision impaired	Age of onset	Mode of inheritance	Bilateral/ Unilateral	Description of the opacity	Abnormal substance(s)
Macular or spotted corneal dystrophy (MCD)	Groenouw Type II, Bücklers Type II, Fehr Spotted Type	All layers of the cornea; later epithelial erosions. Progressive clouding of the cornea, corneal thinning. Deposits in stromal keratocytes, Descemet layer and endothelium	Yes, photophobia, reduced corneal sensitivity	1st decade	AR	B	Small focal, grey-white, stromal opacities. First anterior axial later all through cornea, ground-glass appearance out to the periphery. Guttae on Descemet	Proteoglycan, keratan sulfate in stromal keratocytes and in histiocytes. Can be keratan sulfate negative or keratan sulfate positve.
Marginal crystalline dystrophy	Bietti	Superficial, peripheral stoma	Some reduction	1st decade	AR,AD	B	Punctate, scintillating, golden opacities and map-like lesions	Lipid?
Pellucid marginal degeneration	—	All layers of the cornea, local disruption of Bowman layer	Yes	2nd to 5th decade	S	B	Inferior peripheral thinning	None identified. Degenerative
Posterior amorphous corneal dystrophy	—	Posterior stroma, Descemet membrane. Axial and peripheral	Yes, usually	1st decade	AD	B	Grey-white, sheet-like opacities, edema. Cornea is thinned.	Lipid in keratocytes
Posterior crocodile shagreen	—	Posterior stroma has large opacities	No	Any age	S	B	Greyish, polygonal opacities separated by clear lines	Collagen. Degenerative
Pre-Descemet membrane or deep filiform dystrophy	Maeder and Danis	Deeper layers of the stroma and almost out to the limbus. Keratocyte degeneration occurs. (Compare with cornea farinata.)	No, may be at risk for keratoconus	4th decade	S,AD	B	Tiny, grey, deep, filiform opacities may be mostly axial or may be diffuse across the cornea	Vacuoles in keratocytes contain lipofusin type lipoprotein. Degenerative
Primary gelatinous drop-like corneal dystrophy	—	Epithelium, Bowman layer, stroma	Yes, photophobia, pain	1st to 2nd decade	AR	B	Gelatinous, subepithelial, mulberry-shaped masses in superficial cornea	Subepithelial amyloid. May be degenerative

recurrent epithelial erosions occur even before the appearance of the typical lattice lines. Photophobia is a prominent symptom. After the amyloid accumulation and erosions appear, large, diffuse opacities form in the axial stroma. The epithelial erosions become less frequent and the opacities become denser, extend almost to the corneal periphery and obscure the lattice lines. Both visual acuity and corneal sensitivity are severely decreased.[36,38] The endothelium is not involved.

LCD type II is AD, develops in the third decade of life and is secondary to systemic amyloidosis. This Meretoja or Finland type amyloidosis is associated with progressive peripheral and cranial nerve palsies, blepharochalasis, dry itchy skin and protruding lips. The amyloid deposits in the stroma in type II are usually 30 to 50 μm in diameter.[37] The lattice lines are more peripheral and less numerous, although longer than those in type I.[39] The opacities are radially oriented and extend from the axial region to the limbus.[4,36] Compared to type I, in type II there are fewer disruptions in the Bowman layer by amyloid deposits. Usually, visual acuity is not impaired until later in life. However, patients over 40 years of age may suffer from painful recurrent erosions.[36] Conjunctival deposits of amyloid are prominent in the subepithelial region.[4,37] The risk of open-angle glaucoma seems to be increased.

LCD type III is bilateral, appears to be AR and is unrelated to a systemic disease. It becomes clinically apparent in the seventh to ninth decade at which time it impairs visual acuity, although no recurrent erosions occur. The stromal lattice lines are radially distributed, extend almost from limbus to limbus and are much thicker than those observed in LCD type I or type II.[4,36,37,39] The epithelium is more regular in type III, there are fewer disruptions in the Bowman layer, and the stromal deposits are larger and more numerous. The amorphous amyloid deposits are located midway between the epithelium and the endothelium. Numerous smaller deposits are localized primarily in the anterior two-thirds of the cornea.[37]

Stock et al.[39] report another variation of lattice dystrophy, LCD type IIIA, with histologic and clinical features similar to those of LCD type III; however, there are corneal erosions and the inheritance pattern is AD.

There are several hypotheses as to the source of the amyloid deposits. Rodriques and Krachmer[4] propose that the deposits are related to activation by mononuclear cells or to stromal matrix precursors. Waring et al.[1] offer two main hypotheses for the origin of the amyloid, namely collagen degeneration or keratocyte synthesis.

Lattice corneal dystrophy constitutes about 15% of the corneal dystrophies. To treat the painful recurrent epithelial erosions in types I and II, artificial tears, hypertonic solutions, pressure patching and soft contact lens may be applied. Penetrating keratoplasty is used for all three types of lattice corneal dystrophy; however, for type II it is rarely needed. Unfortunately, lattice dystrophy recurs in grafts (more commonly than recurrences in corneas with macular or granular dystrophy) 2 to 14 years postoperatively.[1,37] See also primary gelatinous drop-like dystroph.

A type of secondary amyloidosis with subepithelial, salmon-coloured, raised deposits can develop in the cornea in congenital glaucoma, keratoconus, leprosy, phlyctenulosis, retinopathy of prematurity, sympathetic ophthalmia or trachoma, or following trauma.[40]

Patients with X-linked ichthyosis can have an anterior stromal dystrophy resembling that of type I lattice dystrophy. A deep punctate dystrophy has also been described in patients with ichthyosis.[16] A polymorphic stromal dystrophy has been described by Pillat in 1939, by Grayson and Wilbrandt,[41] by Strachan in 1968 and by Thomsitt and Bron.[42] The whole group of conditions may be forms of pre-Descemet dystrophy. Flaky, punctate, dendritic, filamentous, boomerang, linear, comma-shaped and circular opacities are reported.[41,42] Most of these opacities are in the posterior stroma with a few in the anterior stroma. Near the limbus, glass-like lines, 0.75 mm long, are in the anterior stroma. The opacities appear in the sixth decade, bilaterally, mostly in the axial stroma and show little progression. An increased prevalence of anterior membrane disorders and keratoconus has been noted in these patients.[41] Corneal sensitivity is normal.

Macular corneal dystrophy

Macular corneal dystrophy (MCD), a bilateral, AR dystrophy, produces progressive clouding of the cornea by irregular corneal opacities. This dystrophy is a relatively uncommon cause of corneal dystrophy except in locations where high rates of consanguinity exist. It has been called Groenouw macular type II or Bücklers type II or Fehr punctate corneal dystrophy. Between five and nine years of age, small white opacities appear in or near the Bowman layer in the axial cornea. About 10 years later, the opacities increase in number and size and spread throughout the entire stroma and peripherally to the limbus (figure 7). The corneal opacities are more anterior in the axial region and more posterior in the corneal periphery. Visual acuity and corneal sensitivity decrease; however, at this time there is marked photophobia but no ocular irritation or inflammation.[1,43–46] By the fourth decade, epithelial erosions tend to occur. Stromal corneal thinning has been documented.[4] The white stromal opacities in MCD exhibit heterogeneity. In this mucopolysaccharidosis at least two distinct varieties of opacities are present: keratan sulfate negative (type 1) and keratan sulfate positive (type 2).[43,44,46] The corneal accumulations consist of either an abnormal keratan sulfate-related molecule or another carbohydrate-rich substance. Keratan sulfate, a glycosaminoglycan, is present in the stroma, keratocytes, Descemet membrane and endothelium.[43,46,47] The keratocytes contain large vacuoles in their cytoplasm. One theory of the cause of macular corneal dystrophy is an inborn deficiency of sulfotransferase which

Figure 7. Macular corneal dystrophy

is needed in the sulfation of the lactosaminoglycan portion of the keratan sulfate chain.[46] This corneal dystrophy is part of a systemic disorder of keratan sulfate biosynthesis. The serum level of keratan sulfate is low.

MCD exhibits three characteristic morphological features: focal, grey-white, stromal opacities; a diffuse stromal haze; and an irregular, cloudy Descemet membrane with corneal guttae.[1] The fine amorphous, granular opacities have indistinct borders and form small, irregular, rounded, grey-white patches. The stromal opacities are most confluent axially and superficially, but gradually extend posteriorly to the endothelium and laterally to the limbus. The result is diffuse, ground-glass, cloudy cornea.

Histologically, the corneal epithelium shows focal areas of thinning and the entire cornea is a little thinner than normal. The glycosaminoglycan is located extracellularly and intracellularly in stromal keratocytes and in endothelial cells. Some areas of the Bowman layer are defective. As MCD progresses, the posterior layer of the Descemet membrane becomes greyer and thicker, while guttate excrescences appear on the endothelium.[1,43] If the endothelial damage results in stromal edema, the cornea will be thicker. The amorphous material in the endothelial cells[43,44] is related to keratan sulfate.

In this relatively common dystrophy, vision is severely impaired by 30 years of age. Symptoms eventually include a foreign body sensation, excessive tearing and photophobia. The most effective treatment for MCD is penetrating keratoplasty, which is required by the third decade. Opacities may recur 1.5 to 11 years postoperatively, but recurrence is uncommon.[1]

Marginal crystalline dystrophy

In 1937 and 1942, Bietti described a marginal crystalline corneal dystrophy involving the superficial layers of the peripheral stroma in two patients with albipunctate dystrophy. Corneal dystrophy can accompany this form of tapetoretinal degeneration or crystalline retinopathy (AR,AD). In Bietti tapetoretinal degeneration, a systemic disorder of lipid metabolism,[48,49] the manifestations are yellow-white crystals in the posterior pole of the fundus, atrophy of the RPE, choroidal sclerosis, poor night vision and abnormal colour vision. Typically, these eyes have reduced vision, field defects and subnormal electrophysiological test results.[50] Some, but not all, patients with Bietti tapetoretinal degeneration have tiny, superficial, mostly peripheral,[50] punctate, golden crystals[48] and map-like lacunae in the corneal stroma which are said to be produced or aggravated by exposure to sunlight (ultraviolet?). The crystals are located in corneal and conjunctival fibroblasts and less frequently in the extracellular space.[49] Compare with climatic droplet dystrophy and with central crystalline dystrophy.

Pellucid marginal degeneration

Pellucid marginal degeneration, formerly called ectatic marginal degeneration, is a bilateral, sporadic, corneal degeneration involving the thinning of the inferior third of the cornea,[12,51,52] one or two millimetres from the limbus and concentric with it. This degeneration occurs in the second to fifth decade of life and produces no scarring, neovascularization or infiltration. The local 1- to 2-mm wide band of thinning reduces the thickness of the cornea in the affected area to 20% of normal, and as the cornea weakens, its local curvature can increase as much as 20 diopters.[34] The area above the band of thinning protrudes anteriorly and remains clear. The thinning causes high, irregular against-the-rule astigmatism; however, corneal sensation remains normal.[34,51,52] Pellucid marginal degeneration is usually diagnosed in males or females in their third or fourth decade. Rodriques et al.[51] and Krachmer et al.[34] suggest that pellucid marginal degeneration may be a form of keratoconus involving the peripheral rather than the central cornea.

Histologically, in the peripheral thinned area the epithelium is irregular in thickness and the Bowman layer is focally disrupted and replaced by irregular connective tissue. The stroma is compressed peripherally, causing it to be markedly thinned. In the superficial stroma, a small number of histocytes are present.[51] Throughout the affected area of the stroma, electron-dense granular deposits and fibrous collagen with a periodicity of 100 to 110 nm (normal periodicity is 60 to 64 nm) are present.[51,52] The Descemet membrane becomes folded or convoluted. The endothelial cells are partially degenerated or absent.[51] Corneal sensitivity is normal. Vascularization does not develop.

Spectacle correction of pellucid marginal degeneration is usually unsatisfactory unless the condition is mild. Hard contact lenses are difficult to fit due to the pronounced inferior steepening. Penetrating keratoplasty may be difficult because of the large eccentric graft needed. One

of the more promising treatments is lamellar keratoplasty.[34,52] Other treatments include diathermy and thermokeratoplasty.[34] Compare with the marginal degeneration of Terrien and with Mooren ulcer. The furrow degeneration and marginal ectasia of Terrien affect the superficial peripheral cornea. See also keratoglobus.

Posterior amorphous corneal dystrophy

Posterior amorphous corneal dystrophy is AD, bilateral and symmetrical. The signs are posterior stromal sheet-like opacities and corneal thinning.[53] Dunn et al.[54] reported that this rare corneal dystrophy may be congenital, because they saw it in a six-month-old child. Typically, posterior amorphous dystrophy becomes apparent in the first decade of life but produces no symptoms. The slowly progressive stromal thinning reduces corneal thickness from the normal 0.5 mm to about 0.3 mm.[1] The thinning leads to other ocular problems including hyperopia, flattened corneal curvature, stromal and anterior iris surface abnormalities, fine iris processes extending to the prominent Schwalbe line and extension of the corneal opacity to the limbus.[4,54] Dunn et al.[54] suggest that a defect in the forward growth or differentiation of embryonic mesoderm could account for the anomalies of the cornea and the iris stroma that occur in posterior amorphous corneal dystrophy. Compare with Axenfeld-Rieger syndrome and similar conditions once called anterior mesenchymal dysgenesis.

The epithelium, basement membrane and Bowman layer are all ultrastructurally normal. The anterior two-thirds of the stroma has normal keratocytes and the architecture of the collagen lamellae is normal. Just anterior to Descemet membrane, the stromal lamellae show various degrees of disorganization. In the most disorganized region, there is mild stromal edema which separates the individual collagen fibres. The keratocytes appear rounded. Large lipid droplets are visible in the cytoplasm. In the stroma, electron-dense granular bodies formed from 10-nm collagen fibres are present in a homogeneous matrix.[55] Discontinuities between the opacities allow clear stroma to be seen.[1,54] Diffuse, grey-white, sheet-like opacities form indistinct patches at various levels across the posterior stroma. In some patients, the opacity extends to the limbus and deepens to involve the Descemet membrane and to disrupt the endothelial mosaic; however, in other patients clear stroma is visible between the opacity and the Descemet membrane. The anterior banded region of the Descemet membrane is interrupted focally by a fibrillar layer that contains 30-nm collagen fibres, similar to the stromal collagen fibres. The endothelium is often normal.[55] Acuity can be normal or reduced to about 6/12.

Corneal thinning is a feature of keratoconus, keratoglobus, macular corneal dystrophy, Mooren ulcer, pellucid marginal degeneration and posterior amorphous corneal dystrophy.[4,28,29,34,53]

Posterior crocodile shagreen

Posterior crocodile shagreen consists of large, asymmetrical, greyish, polygonal, axial opacities that have indistinct borders and are separated by clear lines. This degeneration can occur at any age, although it is generally considered to be an aging change.[14,56] Krachmer et al.[56] state that a hereditary pattern has not been identified for posterior crocodile shagreen. Several authors have reported cases that were bilateral.[3] The opacity is in the posterior two-thirds of the stroma. The density of the lesion increases toward the posterior surface.[56] This condition does not impair vision.[14,56]

Histologically, both the epithelium and the Bowman layer are normal. In the posterior stroma, the collagen lamellae show an irregular sawtooth pattern. Affected areas consist of abnormally arranged collagen fibrils at right angles to each other, and these are dispersed among normal collagen fibrils above and below. Within the abnormal areas, patches of widely spaced (100 nm) collagen fibrils are present.[56] Both the Descemet membrane and the endothelium remain normal.

Treatment is rarely indicated, as posterior crocodile shagreen does not impair the visual acuity.[57] Posterior crocodile shagreen resembles the central cloudy dystrophy of François; however, Ansons and Atkinson[3] point out that crocodile shagreen forms a discrete posterior layer while cloudy dystrophy is ill-defined and extends anteriorly. See also anterior crocodile shagreen.

Posterior crocodile shagreen and, rarely, anterior crocodile shagreen have been reported in patients with lecithin-cholesterol acetylase (LCAT) deficiency, a lipid disorder.[58] In the LCAT disorder,[9] minute, grey-white dots are present throughout the stroma but are most abundant in the pupillary zone. These opacities are present in both eyes from early childhood. The cornea looks dull, especially near the limbus. The opacification is progressive, but corneal thickness remains normal and acuity is little affected. The dots consist of phospholipids and cholesterol. There is a clear space at the limbus but just inside this there are more dots in the anterior stroma. In the periaxial region the dots are more numerous in the posterior stroma. Other corneal layers are normal.

In another disorder of lipid metabolism called fish eye disease,[9] the grey-white or yellowish dots form a mosaic pattern. The lipid dots are in all stromal layers. Vacuoles about 2 μm in diameter are located between the collagen fibres in the stroma, but most of the vacuoles are in the Bowman layer. The cloudy cornea is more opaque toward the periphery. Other corneal layers are normal.

Pre-Descemet membrane or deep filiform corneal dystrophy

Pre-Descemet membrane or the deep filiform corneal dystrophy of Maeder and Danis[59] exhibits numerous tiny opacities located deep in the stroma anterior to the Descemet membrane. The perilimbal cornea is clear. These opacities are usually bilateral, symmetrical and do not reduce visual acuity or produce symptoms. The dots become apparent after 30 years of age, and are assumed to be the result of a degenerative process.[1,13,60] The presence of lipofuscin, a pigment associated with aging, has been identified.[40] The cause of pre-Descemet membrane dystrophy is not entirely certain. Most cases are sporadic, but AD inheritance has been reported. Curran et al.[13] suggest that this dystrophy is a keratocyte degeneration associated with aging. It may be related to cornea farinata,[14] or to the pre-Descemet dystrophy described by Grayson and Wilbrandt[41] or to the polymorphic stromal dystrophy of Thomsitt and Bron.[42] See also fleck dystrophy. A similar dystrophy can be associated with ichthyosis and with pseudoxanthoma elasticum. Dot or comma-shaped pre-Descemet opacities scattered from the axis to the limbus are present in about 25% of patients with ichthyosis and in carriers of this X-linked condition.[61] Franceschetti described this as deep punctate dystrophy. See also central cloudy corneal dystrophy and the deep, axial, polymorphic, parenchymatous dystrophy of Pillat. The amyloid deposits in the Pillat dystrophy probably indicate a type of degeneration.[62] See also the polychromatic dystrophy described by Fernandez-Sasso et al.[63] In this deep stromal dystrophy, punctiform polychromatic filaments occupy the corneal stroma all the way to the limbus. Vision is not affected.[63]

The deep stromal opacities in pre-Descemet dystrophy localize in different areas of the cornea. Some occupy only the axial region, or an annular opacity may form with a clear centre and periphery, while others spread diffusely across the entire cornea. The individual grey opacities assume a variety of shapes such as comma, circular, dendritic, boomerang or worm-shaped.[1,42]

Histologically, the keratocytes deep in the stroma anterior to the Descemet membrane are vacuolated and many contain dense intracytoplasmic inclusions. The vacuoles represent secondary lysosomes that contain phospholipids and unsaturated fats forming residual bodies. The single-membrane-limited vacuoles measure 1 μm in diameter and contain corkscrew-shaped fibrogranular material and electron-dense lamellar bodies. The epithelium, Bowman layer, Descemet membrane and endothelium are all normal histologically and clinically.[13]

Pre-Descemet membrane corneal dystrophy is asymptomatic and does not impair visual acuity,[1] so no treatment is necessary. The prevalence of keratoconus was thought to be increased in these eyes.[59]

Primary gelatinous drop-like corneal dystrophy

Primary gelatinous drop-like dystrophy or familial, subepithelial, amyloid dystrophy is a rare, bilateral, localized corneal form of hereditary amyloidosis (AR) with no systemic associations.[4,64,65] Clinical manifestations of this progressive dystrophy appear between 8 and 18 years of age with photophobia, conjunctival hyperemia, excess lacrimation, a gritty sensation and occasionally blurred vision.[65] In the axial region beneath the corneal epithelium, numerous prominent gelatinous excrescences appear as mulberry-shaped masses.[64,65] The patient with this dystrophy experiences severe vision reduction and some have epithelial erosions. The cause of primary granular corneal dystrophy remains unknown.[65] Rodriques and Krachmer[4] postulate that the amyloid may come from the epithelial cells, keratocytes or macrophages. Primary gelatinous drop-like dystrophy may be a degenerative condition. It is more common in Japan.

Histologically, the epithelium has areas of irregular hyperplasia, areas of atrophy and areas of edema. The epithelial basement membrane is lost. The Bowman layer is destroyed at the sites of the nodes.[4,64] The anterior stroma exhibits fibrosis, neovascularization and numerous superficial deposits of amyloid.[64]

Several types of systemic amyloidosis cause corneal amyloid deposition. See, for example, lattice corneal dystrophy type II.

The treatment for gelatinous drop-like dystrophy is penetrating keratoplasty.[64]

This research was supported in part by the Canadian Optometric Education Trust Fund. The photographs were kindly supplied by these investigators: figure 1, Dr T.D. Williams; figure 2, Dr W.F. Long; figures 3 to 7, Dr A.P. Cullen.

References

1. Waring GO III, Rodriques MM, Laibson PR. Corneal dystrophies, Part I. Dystrophies of the epithelium, Bowman's layer and stroma. Surv Ophthalmol 1978;23:71–122.
2. Bramsen T, Ehlers N, Baggesen LH. Central cloudy corneal dystrophy of François. Acta Ophthalmol 1976;54:221.
3. Ansons AM, Atkinson PL. Corneal mosaic patterns—morphology and epidemiology. Eye 1989;3:811–15.
4. Rodriques MM, Krachmer JH. Recent advances in corneal stromal dystrophies. Cornea 1988;7:19–29.
5. Weller RO, Rodger FC. Crystalline stromal dystrophy: histochemistry and ultrastructure of the cornea. Br J Ophthalmol 1980;64:46–52.
6. Brownstein S, Jackson WB, Onerheim RM. Schnyder's crystalline corneal in association with hyperlipoproteinemia: histopathological and ultrastructural findings. Can J Ophthalmol 1991;26:273–79.
7. Rodriques MM, Kruth HS, Krachmer JH, Vrabec MP, Blanchette-Mackie J. Cholesterol localization in ulterathin frozen sections in Schnyder's corneal crystalline dystrophy. Am J Ophthalmol 1990;110:513–17.

8. Freddo TF, Polack FM, Leibowitz HM. Ultrastructural changes in the posterior layers of the cornea in Schnyder's crystalline dystrophy. Cornea 1989;8:170–77.
9. Barchiesi BJ, Eckel RH, Ellis PP. The cornea and disorders of lipid metabolism. Surv Ophthalmol 1991;36:1–22.
10. Witschel H, Fine BS, Grøtzner P, McTique JW. Congenital hereditary stromal dystrophy of the cornea. Arch Ophthalmol 1978;96:1043–51.
11. Keates RH, Cvintal T. Congenital hereditary corneal dystrophy. Am J Ophthalmol 1965;60:892–94.
12. Friedlaender MH, Smolin G. Corneal degenerations. Ann Ophthalmol 1979;11:1485–95.
13. Curran RE, Kenyon KR, Green WR. Pre-Descemet's membrane corneal dystrophy. Am J Ophthalmol 1974;77:771–76.
14. Sugar A. Corneal and conjunctival degeneration. In: Kaufman HE, Barron BA, McDonald MB, Waltman SR, eds. The Cornea. New York: Churchill Livingstone, 1988:451–52.
15. Grayson M, Wilbrandt H. Pre-Descemet's dystrophy. Am J Ophthalmol 1967;64:276–82.
16. Duke-Elder S, Leigh AG. Diseases of the outer eye. In: Duke-Elder S, ed. System of Ophthalmology. London: Kimpton, 1951:951–52.
17. François J. Dystrophic dermo-chondro-cornéene familiale. Ann Ocul (Paris) 1949;182:409–42.
18. Jensen VJ. Dermo-chondro-corneal dystrophy. Acta Ophthalmol 1958;36:71–78.
19. François J. Heredity in ophthalmology. St Louis: CV Mosby, 1961:310.
20. Nicholson DH, Green WR, Cross HE, Kenyon KR, Massof D. A clinical and histopathological study of François-Neetens speckled corneal dystrophy. Am J Ophthalmol 1977;83:554-60.
21. Purcell JJ, Krachmer JH, Weingeist TA. Fleck corneal dystrophy. Arch Ophthalmol 1977;95:440–44.
22. Patten JT, Hyndiuk RA, Donaldson DD, Herman SJ, Ostler HB. Fleck (mouchetée) dystrophy of the cornea. Ann Ophthalmol 1976;8:25–32.
23. Wittebol-Post D, van der Want JJL, van Bijsterveld OP. Granular dystrophy of the cornea (Groenouw's type I): Is the keratocyte the primary source after all? Ophthalmologica 1987;195:169–77.
24. Ruusuvaara P, Setälä K, Tarkkanen A. Granular corneal dystrophy with early stromal manifestations. Acta Ophthalmol 1990;68:525–31.
25. Møller HV. Inter-familial variability and intra-familial similarities of granular corneal dystrophy Groenouw type 1 with respect to biomicroscopical appearance and symptomatology. Acta Ophthalmol 1989;67:669–77.
26. Møller HV. Granular corneal dystrophy. Groenouw type I: Clinical aspects and treatment. Acta Ophthalmol 1990;68:384–49.
27. Waardenburg PJ, Jonkers GH. A specific type of dominant progressive dystrophy of the cornea, developing after birth. Act Ophthalmol 1961;39:919–23.
28. Lawless M, Coster DJ, Phillips AJ, Loane M. Keratoconus: diagnosis and management. Aust NZ J Ophthalmol 1989;17:33–60.
29. Bron AJ. Keratoconus. Cornea 1988;7:163–69.
30. Lembach RG. Keratoconus. Int Ophthalmol Clin 1991;31:71–82.
31. Perry HD, Buxton JN, Fine BS. Round and oval cones in keratoconus. Ophthalmology 1980;87:905–9.
32. Stern GA, Knapp A, Hood CI. Corneal amyloidosis associated with keratoconus. Ophthalmology 1988;95:52–55.
33. McPherson SD, Kiffney GP. Some histologic findings in keratoconus. Arch Ophthalmol 1968;79:669–73.
34. Krachmer JH, Feder RS, Belin MW. Keratoconus and related noninflammatory corneal thinning disorders. Surv Ophthalmol 1984;28:293–322.
35. Dubord PJ, Krachmer JH. Diagnosis of early lattice corneal dystrophy. Arch Ophthalmol 1982;100:788–90.
36. Hida T, Tsubota K, Kigasawa K, Murata H, Ogata T, Akiya S. Clinical features of a newly recognized type of lattice corneal dystrophy. Am J Ophthalmol 1987;104:241–48.
37. Hida T, Proia AD, Kigasawa K, et al. Histopatholgic and immunochemical features of lattice corneal dystrophy type III. Am J Ophthalmol 1987;104:249–54.
38. Tsubota K, Hida T, Murata H, Akiya S, Sinji T, Kimura W. Lattice dystrophy type I: A report of 8 families. Ophthalmologica 1987;194:71–76.
39. Stock EL, Feder RS, O'Grady RB, Sugar J, Roth SI. Lattice corneal dystrophy type IIIA. Arch Ophthalmol 1991; 109:354–58.
40. Smolin G. Dystrophies and Degenerations. In: Smolin S, Thoft RA, eds. The Cornea: Scientific Foundations and Clinical Practice. Boston, Little Brown and Company, 1987:427–55.
41. Grayson M, Wilbrandt H. Pre-Descemet dystrophy. Am J Ophthalmol 1967;64:276–82.
42. Thomsitt J, Bron AJ. Polymorphic stromal dystrophy. Br J Ophthalmol 1975;59:125–32.
43. Johnasson F, Johannsson JH, Garner A, Rice NSC. Macular corneal dystrophy in Iceland. Eye 19889;3:446–54.
44. Edward DP, Yue BYJT, Sugar J, et al. Heterogeneity in macular corneal dystrophy. Arch Ophthalmol 1988; 106:1579–83.
45. Meek KM, Quantock AJ, Elliott GF, et al. Macular corneal dystrophy: the macromolecular structure of the stroma observed using electron microscopy and synchrotron x-ray diffraction. Exp Eye Res 1989;49:941–58.
46. Yang W, SundarRaj N, Thonar EJ-MA, Klintworth GK. Immunohistochemical evidence of heterogeneity in macular corneal dystrophy. Am J Ophthalmol 1988;106:65–71.
47. Edward DP, Thonar EJ-MA, Srinivasan M, Yue BJYT, Tso MOM. Macular dystrophy of the cornea: a systemic disorder of keratan sulfate metabolism. Ophthalmology 1990;97:1194–200.
48. Richards BW, Brodstein DE, Nussbaum JJ, Ferencz JR, Maeda K, Weiss L. Autosomal dominant crystalline dystrophy. Ophthalmology 1991;98:658–65.
49. Wilson DJ, Weleber RG, Klein ML, Welch RB, Green WR. Bietti's crystalline dystrophy, a clinicopathologic correlative study. Arch Ophthalmol 1989;107:213–21.
50. Harrison RJ, Acheson RR, Dean-Hart JC. Bietti's tapetoretinal degeneration with marginal corneal dystrophy (crystalline retinopathy): case report. Br J Ophthalmol 1987;71:220–23.
51. Rodriques MM, Newsome DA, Krachmer JH, Eiferman RA. Pellucid marginal corneal degeneration: a clinopathologic study of two cases. Exp Eye Res 1981;31:277–88.
52. Deluise VP. Peripheral corneal degenerations and tumors. Int Ophthalmol Clin 1986;26:49–61.
53. Carpel EF, Sigelman RJ, Doughman DJ. Posterior amorphous corneal dystrophy. Am J Ophthalmol 1977; 83:629–32.
54. Dunn SP, Krachmer JH, Ching SST. New findings in posterior amorphous corneal dystrophy. Arch Ophthalmol 1984;102:236–39.
55. Johnson AT, Folberg R, Vrabec MP, Florakis GJ, Stone EM, Krachmer JH. The pathology of posterior amorphous corneal dystrophy. Ophthalmology 1990;97:104–9.
56. Krachmer JH, Dubord PJ, Rodriques MM, Mannis MJ. Corneal posterior crocodile shagreen and polymorphic amyloid degeneration: a histopathologic study. Arch

Ophthalmol 1983;101:54–59.
57. Sugar A. Corneal and conjunctival degenerations. In: Kaufman HE, Barron BA, McDonald MB, Waltman SR, eds. The Cornea. New York: Churchill-Livingstone, 1988:451–2.
58. Vrabec MP, Shapiro MB, Koller E, Wiebe DA, Henricks J, Albers JJ. Ophthalmic observations in lecithin cholesterol acyltransferase deficiency. Arch Ophthalmol 1988; 106:225–29.
59. Maeder G, Danis P. Sur une nouvelle forme de dystrophie cornéene (dystrophia filiformis profunda corneae) associée à un kératocône. Ophthalmologica 1947;114:246–48.
60. Dodwell DG, Freeman K, Shoch D. Juvenile asteroid hyalosis and pre-Descemet's dystrophy. Am J Ophthalmol 1988;106:504–5.
61. Costagliola C, Fabbrocini G, Illiano GMP, Scibelli G, Delfino M. Ocular findings in x-linked ichthyosis: a survey of 38 cases. Ophthalmologica 1991;202:152–55.
62. François J. Heredo-familial corneal dystrophies. Trans Ophthalmol Soc UK 1966;86:367–416.
63. Fernandez-Sasso D, Acosta JEP, Malbran E. Punctiform and polychromatic pre-Descemet's dominant corneal dystrophy. Br J Ophthalmol 1979;63:336–38.
64. Gartry DS, Falcon MG, Cox RW. Primary gelatinous drop-like keratopathy. Br J Ophthalmol 1989;73:661–64.
65. Akiya S, Furukawa H, Sakamoto H, Takahashi H, Sakka Y. Histopathologic and immunohistochemical findings in gelatinous drop-like corneal dystrophy. Ophthalmic Res 1990;22:371–76.

How useful is the VisTech MCT8000 for contrast sensitivity and glare testing?

David B Elliott

key words: Vistech MCT8000, contrast sensitivity, disability glare, repeatability

mots-clés : Vistech MCT8000, sensibilité différentielle, éblouissement perturbateur, répétabilité

This study evaluated the usefulness of the Vistech MCT8000 CS and disability glare measurements by their reliability and redundancy. Three subject groups were used: young normal (N=24), older normal (N=22) and cataract (N=32). The high correlations between adjacent spatial frequency CS scores suggest that much of the information collected is superfluous. All that is needed is a reliable VA measurement and an assessment of CS at or near the peak of the CS curve. Test–retest results of 37 subjects indicate the MCT results have poor repeatability. The important factors are the step size and the psychophysical method of threshold determination. The standardization of the glare source appear as to be of less importance if these factors are not optimised.

L'étude actuelle évalue l'utilité dès mesures de la SD et de l'éblouissement perturbateur à l'aide du Vistech MCT8000, en se fondant sur leur fiabilité et leur redondance. Trois groupes témoin ont été utilisés : jeune normal (N=24), vieux normal (N=22) et cataracte (N=32). Les corrélations élevées entre les notes adjacentes de SD de fréquence spatiale indiquent que la plus grande partie de l'information recueillie est superflue. Tout ce dont on a besoin est une mesure fiable de l'acuité visuelle et une évaluation de la SD au sommet ou près du sommet de la courbe de SD. Les résultats de tests indiquent une piètre répétabilité des résultats à l'aide du MCT. La taille de l'incrément et la méthode psycho-physique de détermination du seuil constituent des facteurs importants. La normalisation de la source d'éblouissement semblent moins importantes lorsque de tels facteurs ne sont pas optimisés.

DB Elliott, PhD, MBCO—Centre for Sight Enhancement, School of Optometry, University of Waterloo

The clinical applications of contrast sensitivity (CS) measurements may not be as extensive as was at one time hoped. They have been shown to be particularly useful, however, in evaluating optic neuropathy, in explaining symptoms of poor vision in patients with good visual acuity (VA) and in providing a more comprehensive evaluation of visual function in low vision patients, particularly those with cataract.[1,2] Disability glare measurements are also becoming more accepted as a useful clinical tool in the evaluation of patients with media opacities, and in the evaluation of cataract and refractive surgery.[3] This is reflected by the increased use of CS and glare testing in clinical practice. For example, in a recent survey of 396 ophthalmologists of the American Society of Cataract and Refractive Surgery, 65% of respondents were routinely using CS, glare testing or both to evaluate visual function in cataract.[4] This has led to a proliferation of proposed techniques and commercially available tests.[3] Of those ophthalmologists who reported using CS charts,[4] 79% were using Vistech charts.[5] More recently, Vistech have introduced the Multivision Contrast Tester (MCT 8000). The present study evaluates the usefulness of the Vistech MCT8000 CS and disability glare measurements by their reliability and redundancy.

Subjects

Subjects were recruited from a local ophthalmologist's office and from the staff, student and patient population of the School of Optometry, University of Waterloo. Three subject groups were recruited: young normal (N=24, mean age 24.3 ± 3.3 years), older normal (N=22, mean age 66.0 ± 6.2 years) and early cataract (N=32, mean age 70.6 ± 8.1 years). Subjects with poor general health, diabetes, a refractive error greater that ±6.00DS, any history of amblyopia or ophthalmic surgery were excluded. To ensure that any loss of visual function was due to lens changes, subjects with intraocular pressure greater that 21 mmHg or with any ocular disease other than cataract were excluded. All cataract and older normal subjects had been screened for ocular disease by ophthalmoscopy, slit-lamp biomicroscopy and applanation tonometry. Cataract subjects had visual acuities of 6/18 or better and normal subjects had visual acuities of 6/7.5 or better.

Apparatus

The targets and glare sources of Vistech MCT8000 are all contained within a portable unit. A console provides control of target presentation and luminance and glare source position. Calibrating the chart and glare source luminance levels is quick and easy. The unit allows measurement of CS at 1.5, 3, 6, 12 and 18 c/deg under

"nighttime" (3 cd/m^2) and "daytime" (125 cd/m^2) luminance conditions with or without a central or peripheral glare source. It is suggested that the central glare source be used under nighttime luminance to simulate night driving conditions, and the peripheral glare source be used under daytime luminance to simulate bright sunlight.[6] The peripheral glare source consists of an oval of small lights in front of both eyes, about half of them visible with the eye tested. Each target consists of seven circular discs, each containing a sine-wave grating of a fixed spatial frequency. The gratings are either vertical or tilted 15° to the right or left, and the contrast of the gratings progressively decreases from disc one to disc seven. Starting at disc one, the subject is asked to indicate the orientation of each grating, or to respond "blank" if nothing is seen. The last disc whose orientation is correctly identified represents the CS score. Measurement of CS at 6 c/deg with and without glare is described as a "functional disability test" (FDT) and is proposed as an initial screening technique.[6] The instrument also allows the measurement of potential acuity and photostress recovery time.

Method

The manufacturer's recommended testing procedures were adopted. All measurements were made monocularly in a dimly lit room, using natural pupils and appropriate optical refraction. FDT under nighttime illumination with and without a central glare source and all five spatial frequencies under daytime conditions with and without peripheral glare were measured. Due to time limitations, only FDT measurements were made in 19 of the cataract subjects. As recommended,[6] nighttime measurements were always made first. The order of daytime measurements was randomised, and all subjects were allowed recovery from any glare source afterimage before starting the next test. The chart and glare source luminance levels were checked using the internal calibration feature before taking measurements for each subject.

LogMAR VA measurements were made with the Regan 96% contrast chart at a luminance of 100 cdm-2.

Measurements from 37 subjects (19 young normal, 11 older normal, 7 cataract) were repeated under identical conditions between 1 and 12 weeks later.

Results

Mean logMAR VA for the young normal, older normal and cataract groups were -0.20 (~6/4), -0.10 (~6/5) and 0.17 (~6/9) respectively. Mean (±1 sd) values for all CS measurements are shown in table 1. Values are given for with and without the glare source. The mean daytime CS values at each spatial frequency for the three subject groups are shown in figure 1. The results suggest there is a difference in log CS between the young and older normal subjects at the higher spatial frequencies. A two-factor ANOVA confirms this, with significant differences in log CS with spatial frequency ($F_{4,44}$=115.7, p<<0.001), age group ($F_{1,44}$=9.3, p<0.005) and a significant interaction effect ($F_{4,44}$=8.2, p<0.001). This difference is increased when CS is measured with the peripheral glare source. There is also a highly significant difference between the scores for the two age groups on the nighttime FDT (6 c/deg) test for both central glare (t_{45}=–9.8, p<<0.001) and no glare conditions (t_{45}=-8.3,

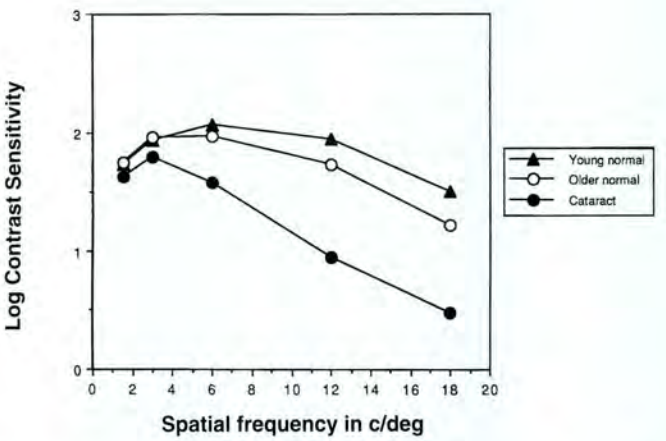


Figure 1. The mean daytime log CS values at each spatial frequency for the three subject groups (young normal, N=24; older normal, N=22; cataract, N=13).

Table 1. Mean values of log CS

Subject group	Glare	Nighttime luminance 6c/deg	Daytime luminance 1.5 c/deg	3 c/deg	6 c/deg	12 c/deg	18 c/deg
Young	no	1.81±0.15	1.74±0.16	1.94±0.10	2.08±0.07	1.95±0.09	1.51±0.21
	yes	1.60±0.24	1.68±0.17	1.93±0.12	2.07±0.09	1.94±0.08	1.51±0.24
Older	no	1.61±0.18	1.75±0.20	1.97±0.14	1.99±0.14	1.74±0.31	1.22±0.32
	yes	1.23±0.28	1.71±0.21	1.95±0.19	1.94±0.15	1.58±0.37	1.00±0.47
Cataract	no	1.19±0.27	1.63±0.15	1.81±0.15	1.59±0.23	0.95±0.37*	(N=8)
	yes	(N=6)	1.43±0.29	1.59±0.24	1.47±0.26	(N=7)	(N=4)

Mean (±sd) values of log CS under nighttime (at 6c/deg) and daytime luminances (at all five spatial frequencies) for the three subject groups of young normal (N=24), older normal (N=22) and cataract (daytime measurements, N=13; nighttime, N=32). A central glare source was used with nighttime luminance conditions and a peripheral glare source under daytime conditions.
*N=12

Table 2. COR log CS values

	Nighttime luminance			Daytime luminance		
Glare	6c/deg	1.5 c/deg	3 c/deg	6 c/deg	12 c/deg	18 c/deg
no	±0.48[a]	±0.47	±0.41	±0.30	±0.49	±0.45[b]
yes	±0.66[d]	±0.51	±0.45	±0.38	±0.66[a]	±0.59[c]

Coeffefficient of repeatability (COR) log CS values from test–retest data of 37 subjects (19 young normal, 11 older normal and 7 cataract).
[a]N=35
[b]N=33
[c]N=32
[d]N=29

Table 3. Correlation matrix of contrast sensitivity

	1.5 c/deg	3 c/deg	6 c/deg	12 c/deg	18 c/deg	VA
1.5 c/deg	1.0					
3 c/deg	0.35	1.0				
6 c/deg	0.33	0.45	1.0			
12 c/deg	0.25	0.42	0.80	1.0		
18 c/deg	0.23	0.32	0.60	0.73	1.0	
VA	–0.20	–0.37	–0.66	–0.75	–0.60	1.0

Correlation matrix of contrast sensitivity at each spatial frequency of the Vistech MCT and LogMAR visual acuity from 54 subjects (24 young normal, 22 older normal and 8 cataract).

$p<<0.001$). CS is reduced further in the cataract subjects, particularly at high spatial frequencies (fig 1), and in the presence of a glare source (table 1).

Repeatability

The test-retest interval ranged between 1 and 12 weeks with a median of two weeks. One-tailed t-tests showed no significant difference between test and retest data ($p>0.5$). The coefficients of repeatability (COR)[7] of the test–retest data are shown in table 2. The COR is calculated as 1.96 multiplied by the standard deviation of the differences between test and retest data.

Inter-spatial frequency correlations

Other clinical tests of CS such as the Cambridge[8] and Pelli-Robson[9] charts make one measurement of CS and are designed to be used with a VA measurement. The rationale behind this design is that if VA measurements provide information about the high spatial frequency portion of the CS curve, then only one other measurement of CS is needed at or near the peak of the curve. The Vistech MCT on the other hand makes CS measurements at five points along the CS function. If the design principle of the Cambridge and Pelli-Robson charts is correct, then some of the information provided by the Vistech must be superfluous and therefore represent a waste of chair time. This situation can be assessed by examining the correlation matrix between CS at the five different spatial frequencies on the chart (and also with VA) as shown in table 3. The matrix demonstrates that adjacent spatial frequencies on the MCT are reasonably well correlated with one another, particularly at high spatial frequencies. In this respect, even correlation coefficients between test and retest data of the same spatial frequency tend not to be high because of the test's poor repeatability.[10–15] The high correlation between adjacent spatial frequencies has been demonstrated previously with the Vistech 6500[12,13,16] and indicates a high level of redundant information in the data. Indeed, Kennedy and Dunlap[13] have suggested that the information provided by the five spatial frequencies of the Vistech 6500 can safely be summarized by two scores. One score indicates sensitivity at low spatial frequency and the other at high. The statistical analysis used by Kennedy and Dunlap was repeated and, in addition, the results of logMAR VA were included. A factor analysis of the correlations in table 3 (principle components, normalised by varimax rotation) is shown in table 4. The values presented are correlation coefficients between both logMAR VA and Vistech CS values at each spatial frequency and the two factors revealed by factor analysis. The two lowest spatial frequencies are correlated mainly with factor 2 (the low frequency factor). The highest three spatial frequencies and VA are correlated primarily with factor 1 (the high frequency factor). Before varimax rotation, the percentage variance accounted for by factors 1 and 2 were 50% and an additional 25% respectively. The results of the same analysis of the daytime CS with peripheral glare results and VA were similar and are not shown.

Discussion

The mean and 90% confidence limits of the normal data closely match the normal data provided by the manufac-

Table 4. Varimax orthogonal transformation factor analysis of the correlation matrix

	Factor 1	Factor 2
1.5 c/deg	0.06	0.89
3 c/deg	0.34	0.69
6 c/deg	0.81	0.33
12 c/deg	0.91	0.20
18 c/deg	0.82	0.13
logMAR VA	–0.85	–0.14

turer.[6] Two exceptions are the nighttime FDT scores, where the older normals were about one step lower than the manufacturer's data. This may indicate that subjects were not given sufficient adaptation time. There was a significant difference in log CS between the young and older normal subject groups under daytime luminance at medium and high spatial frequencies (fig 1). This was accentuated by the addition of a glare source. This is not surprising as it confirms many previous findings with CRT and oscilloscope output systems.[17–20] The normative data provided with the Vistech MCT8000, however, combine results from all age groups.[6]

Twenty-six of the 32 cataract subjects could not see the gratings on the nighttime FDT with central glare test. As all the older normal subjects could see the gratings, this indicates that the test discriminates well between cataract and normal. Although the ability to see a target under these conditions could be used as an indicator for the presence of early cataract, it is probably not useful as an indicator of the need for surgery. Only 9 subjects were waiting for surgery, and the majority of the subjects had very early cataract (mean VA 0.17 ± 0.14, Snellen $6/9^{+2}$).

The COR values in table 2 indicate that the MCT results have poor repeatability. COR values range from ±0.30 to ±0.49 log CS under daytime luminance, ±0.38 to ±0.66 log CS under daytime luminance with peripheral glare and ±0.66 and ±0.48 log CS for nighttime FDT scores with and without central glare respectively. For comparison, Pelli-Robson CS[9] COR results with the Brightness Acuity Tester (BAT) glare source, for the same 37 subjects, were ±0.21 log CS. Poor repeatability of results from the Vistech 6500 have also been reported by several other investigators.[10–14] The unreliable nature of the results are probably a combination of poor psychophysical method, single trials at each contrast level and large contrast increments. The Vistech test uses a non-forced choice criterion-dependent psychophysical technique as it allows subjects to make a "blank" response. Criterion-dependent methods have been shown to be less reliable than forced-choice ones.[21] Furthermore, Bailey and coworkers[22] have demonstrated that test reliability is adversely affected by having a test or scale with large step sizes. The Vistech test has only 6 contrast values compared to the Pelli-Robson's 16. Finer steps are available for the Pelli-Robson chart by scoring by letter.[23] The result of having a small number of steps on the Vistech test is that the step sizes are large, with an average of around 0.25 log CS units. This compares with increments of 0.05 log units for the Pelli-Robson chart, when scored by letter. Other than with the central glare source there is little difference between the COR values with and without glare (table 2). The effect of the central glare source may be very position sensitive and adaptation sensitive. The glare sources are well standardised and calibration is quick and easy, so they may appear an unlikely source of any additional variability. Changes in eye position, however, can significantly alter the amount of glare that is received.

The results of the correlation matrix shown in table 3 and the factor analysis results in table 4 are similar to those of previous studies using the Vistech 6500.[13,16] This analysis suggests that CS using the Vistech MCT can safely be summarised by two scores rather than five. The first indicates CS at high spatial frequencies and the other at low. The results further suggest that logMAR VA measurements give a good indication of the high spatial frequency results. VA measurements should be preferred to high frequency Vistech CS results, as they have been shown to be much more reliable,[14] and high spatial frequency results cannot always be obtained with the Vistech. In this study results at 18 c/deg could not be obtained in 9 of the 13 cataract subjects (five without glare) whose VAs were all better than $6/9^{-1}$. Results at both 12 and 18 c/deg could not be obtained in six subjects (one without glare) with VA's better than $6/12^{+4}$. This assessment of the results from the Vistech MCT suggests that the rationale behind the design of the Cambridge and Pelli-Robson charts is correct. Since VA measurements provide reliable information regarding the high spatial frequency end of the CS curve, then only one other measurement is needed at a low to medium spatial frequency.

Conclusion

The high correlations between adjacent spatial frequency CS scores suggest that much of the information collected by the Vistech MCT is superfluous. All that is needed is a reliable VA measurement and an assessment of CS at or near the peak of the CS curve (tables 3 and 4). Other studies have shown that the Vistech 6500 CS charts are less reliable than more recently available charts, particularly those using letter targets.[10–15] This study suggests that this is also true of both the CS and disability glare results from the Vistech MCT8000. The important factors in the reliability and sensitivity of CS and disability glare tests appear to be the increment or step size[3,22,23] and the psychophysical method of threshold determination.[3] The geometry and standardization of the glare source appear to be of less importance if these factors are not optimised.

The Vistech MCT8000 was bought using a COETF grant to Dr John Peroff. I would like to thank Dr Peroff for the loan of the instrument, Drs Hugh Jellie and Dante Pokrnich for providing cataract subjects and Drs Mark Bullimore and Graham Strong for helpful comments of an earlier draft of this report. Financial support was provided by Bausch and Lomb (Canada) and the Centre for Contact Lens Research (CCLR), University of Waterloo.

References

1. Arden GB. Testing contrast sensitivity in clinical practice, Clin Vis Sci 1988:2;213–24.
2. Elliott DB, Whitaker D. How useful are contrast sensitivity measurements in optometric practice? Optom Vis Sci 1992:69;378–85.
3. American Acadamy of Ophthalmology. Contrast sensitivity and glare testing in the evaluation of anterior segment disease. Ophthalmology 1990:97;1233–37.
4. Koch DD, Liu JF. Survey of the clinical use of glare and contrast sensitivity testing. J Cataract Refract Surg 1990:16;707–11.
5. Ginsburg AP. A new contrast sensitivity vision test chart. Am J Optom Physiol Opt 1984:61;403–7.
6. Vistech Consultants, Inc. Multivision contrast tester (MCT8000). Instruction manual.
7. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986:1;307–10.
8. Wilkins AJ, Della Sala S, Somazzi L, Nimmo-Smith I. Age-related norms for the Cambridge low contrast gratings, including details concerning their design and use. Clin Vis Sci 1988:2;201–12.
9. Pelli DG, Robson JG, Wilkins AJ. The design of a new letter chart for measuring contrast sensitivity. Clin Vis Sci 1988:2;187–99.
10. Long GM, Tuck JP. Reliabilities of alternate measures of contrast sensitivity functions. Am J Optom Physiol Opt 1988:65;37–48.
11. Rubin GS. Reliability and sensitivity of clinical contrast sensitivity tests. Clin Vis Sci 1988:2;169–77.
12. Brown B, Lovie-Kitchen JE. High and low contrast acuity and clinical contrast sensitivity tested in a normal population. Optom Vis Sci 1989:66;467–73.
13. Kennedy RS, Dunlap WP. Assessment of the Vistech contrast sensitivity test for repeated-measures applications. Optom Vis Sci 1990:67;248–51.
14. Bradley A, Hook J, Haeseker J. A comparison of clinical acuity and contrast sensitivity charts: effect of uncorrected myopia. Ophthal Physiol Opt 1991:11;218–26.
15. Reeves BC, Wood JM, Hill AR. Vistech VCTS 6500 charts—Within- and between-session reliability. Optom Vis Sci 1991:68;728–37.
16. Elliott DB, Whitaker D. Clinical contrast sensitivity chart evaluation. Ophthal Physiol Opt 1992:12;275-80.
17. Owsley C, Sekuler R, Siemsen D. Contrast sensitivity throughout adulthood. Vis Res 1983:23;689–99.
18. Elliott DB. Contrast sensitivity decline with aging: A neural or optical phenomenon? Ophthal Physiol Opt 1987:7;415–19.
19. Higgins KE, Jaffe MJ, Caruso RC, DeMonasterio FM. Spatial contrast sensitivity: Effects of age, test–retest and psychophysical method. JOSA (A) 1988:5;2173–80.
20. Sloane ME, Owsley C, Jackson CA. Aging and luminance-adaptation effects on spatial contrast sensitivity. JOSA (A) 1988:5;2181–90.
21. Vaegan, Halliday BL. A forced-choice test improves clinical contrast sensitivity testing. Br J Ophthalmol 1982:66;477–91.
22. Bailey IL, Bullimor MA, Raasch TW, Taylor HR. Clinical grading and the effects of scaling. Invest Ophthalmol Vis Sci 1991:32;422–32.
23. Elliott DB, Bullimore MA, Bailey IL. Improving the reliability of the Pelli-Robson contrast sensitivity test. Clin Vis Sci 1991:6;471–75.

Clinical Diagnosis (from page 178)

Diagnosis

The presumptive diagnosis is dominant drusen (Doyne's honeycomb dystrophy).

Drusen are depositions of mucopolysaccharide and lipid substances, originating from degenerating retinal pigment epithelium (RPE) and located between the RPE and Bruch's membrane of the choroid. They result from abnormal lysosomal activity. They are a common, age-related fundus change, occurring in 25% of the population over the age of 52 years. Drusen is rarely present before the age of 45 years.

The observations of drusen in the fundi of this 33-year-old patient is, therefore, significant, and indicates an inherited condition. This should be confirmed by examination of other family members to establish a familial pattern. Unfortunately, this has not yet been possible in DR's case.

Dominant drusen are inherited in an autosomal dominant pattern, with variable penetrance and expressivity. Intra- and inter- familial differences in ophthalmoscopic appearance are common. The drusen are rarely observed before the third decade. They are initially present within the macular region, and occasionally in clusters throughout the posterior pole. Fluorescein angiography shows staining of the drusen. The patient is generally asymptomatic. DR is considered to have dominant drusen at this first stage. Her symptom of vertical diplopia when reading is attributed to uncorrected with-the-rule astigmatism, and the slight decrease in acuity OD is due to refractive amblyopia.

The rate of progression of the condition is unknown. An increase in the size and number of drusen may occur within the fourth decade. This is accompanied by atrophy and pigment clumping of the overlying RPE. Symptoms of mild distortion, blurring of vision or both may be experienced.

More advanced stages of the condition may occur by the fifth to sixth decades. In the atrophic, or dry, form, RPE atrophy leads to atrophy of the overlying photoreceptors, with hyper- and hypopigmentary changes at the macula. Vision is reduced, in the range of 20/60 to 20/80. In the exudative, or wet, form, choroidal neovascularization occurs, associated with haemorrhaging, exudation and elevation of the sensory retina. Fluorescein angiography shows leakage of sub-retinal fluid. Metamorphopsia and decreased vision are experienced. Retinal/RPE detachments, disciform maculopathy or both may result. Final acuity can vary from 20/30 to 20/200. At this stage, argon laser photocoagulation is considered if the neovascular membrane is greater than 200 µm from the foveal avascular zone.

The ERG remains normal throughout the progression of the condition. There are reports of normal and abnormal EOG findings; therefore this test is not considered to have a valuable role in diagnosis or management of dominant drusen.

Management

Since the progression of the condition varies in individual cases, prophylactic laser treatment is not indicated. In fact, there are reported cases of choroidal neovascular membranes occurring in treated eyes. Management therefore involves monitoring of macular function; home use of an Amsler grid is recommended. Low vision aids may be indicated if untreatable vision loss occurs. Genetic counselling is appropriate at all stages of the condition.

References

Alexander LJ. Primary care of the posterior segment. East Norwalk, CT: Appleton & Lange, 1989.

Newsome DA. Retinal dystrophies and degenerations. New York: Raven Press, 1988.

Editor's Note: *We extend an open invitation to any OD wishing to share a particularly interesting case with the readers of the* CJO*RCO *to submit it. Photos are appreciated, but are not essential if the text is sufficiently descriptive. (Our preferred format is a 35mm colour or b&w slide, but a clear, glossy print is also acceptable.) Please send your diagnosis to MJ Samek, OD, FAAO, Senior Editor,* CJO*RCO, *c/o Canadian Association of Optometrists, Suite 301, 1785 Alta Vista Drive, Ottawa, ON, K1G 3Y6.*

Acquired sixth nerve palsy

Maria Marconi

DB, a 51-year-old Caucasian female, was referred for a binocular vision assessment in July 1992. Her chief visual complaint was intermittent horizontal diplopia at distance only, which began one month earlier. No other symptoms were reported and no medications were being taken. A recent physical examination had revealed no health problems. DB reported that she had been in a car accident five years earlier, which resulted in nerve damage to her left arm and hand. She experienced similar diplopia subsequent to her accident for two to three months, after which time the diplopia resolved.

Presenting visual acuities with DB's current spectacle prescription of OD +1.75 DS, OS +1.00 DS (+1.50 Add) were as follows:

At distance:	OD	$6/4.5^{-1}$	At near:	0.37 M
	OS	6/6		0.37 M
	OU	$6/6^{+}$		0.37 M

Cover test revealed an incomitant 18 p.d. alternating esotropia at distance with 2 p.d. esophoria and 7 p.d. right hyperphoria at near. DB reported increased diplopia on left gaze, suggesting a restriction of abduction of the left eye; however, versions and ductions were observed to be full. Uncrossed diplopia was present at distance and near with the Worth Four Dot test. The potential for fusion was confirmed by observation of a fused response on the Worth Four Dot test through 18 p.d. base out/OS.

Confrontation fields were within normal limits, as were other ocular health findings.

DB was diagnosed with recurrent left sixth nerve palsy most likely due to the trauma experienced five years earlier. A 10 p.d. base out Fresnel prism was placed on the left lens as this was the lowest amount of prism through which fusion could be attained. Anti-suppression and anaglyphic fusion training were prescribed.

On her reassessment two weeks later, DB reported no diplopia through the Fresnel prism except on left gaze. Cover test revealed 6 p.d. esophoria without Fresnel prism, at distance only. No hyperphoria was apparent at this visit, therefore, the transient hyperphoria was attributed to the acute left sixth nerve palsy. Stereoacuity was 80 seconds of arc at near, and none was present at distance. DB was counselled to wear the spectacles with the Fresnel prism for driving only, to ensure single vision. Fusion exercises were continued.

At follow-up two weeks later, single vision, both with and without the aid of the Fresnel prism, was reported, except on extreme left gaze. Cover test revealed 2 p.d. esophoria at distance and near. Sixty seconds of stereoacuity was present at near and none was present at distance. The Fresnel prism was removed from DB's spectacles. A free fusion card was prescribed in an effort to promote stereoacuity.

M Marconi, BSc, Optometry Student
School of Optometry, University of Waterloo

Discussion

The typical case presentation of acquired sixth nerve palsy is similar to the case described in that sudden onset diplopia is present at distance only and there is restricted abduction on lateral gaze. Sixth nerve palsy can also present as divergence paralysis. Divergence paralysis has been defined as a comitant esotropia with uncrossed diplopia at distance and orthophoria and fusion at near with full ocular motility.[1] Such cases can develop into a sixth nerve palsy (usually bilateral), however, the mechanism involved has yet to be determined.

Etiology

Although 30% of cases of sixth nerve palsies have an undetermined cause, documented cases have pointed to vascular causes (diabetes, hypertension, aneurysm and atherosclerosis), trauma, neoplasm and complications of lumbar puncture and water-soluble contrast myelography.

Vascular problems account for 17% of sixth nerve palsies. Ischemia due to atherosclerosis or aneurysm of the basilar artery or the internal carotid artery may explain the onset of sixth nerve palsy, especially in older patients.[2,3] Although vascular complications are suspected in cases of sixth nerve palsy associated with diabetes, the mechanism has not yet been determined.

Trauma accounts for 16% of sixth nerve palsies. The most likely mechanism and the one thought to be responsible for the case presented is a blow to the back of the head causing the brain to drop into the foramen magnum and forcing the abducens nerve against the petrosphenoidal ligament. Confusion or avulsion of the nerve could result.[4,5]

Neoplasms account for 14% of sixth nerve palsies in adults. There are three areas along the abducens nerve most susceptible to damage: (1) at the pons-medulla junction where increased intracranial pressure due to a

tumour could displace the brain stem and result in compression of the abducens nerve by the anterior inferior cerebellar artery or internal auditory artery; (2) at the right angle bend made by the abducens nerve upon entering the dura where increased intracranial pressure or direct tumour compression could compromise the abducens nerve directly, or the vascular supply to the nerve; (3) within the cavernous sinus where invasion by nasopharyngeal tumours is possible.[6] It becomes especially important to consider the possibility of neoplasm in children since 40% of acquired sixth nerve palsies in children are due to tumour.[2] Tumour should be suspected if other neurologic signs or symptoms are present such as Horner's syndrome, third/fourth nerve paresis, hypalgesia, retro-orbital pain, papilloedema or Gradenigo's syndrome (otitis media with sixth nerve palsy).[2,6,7]

Although infrequent, sixth nerve palsy may result as a complication of lumbar puncture or water-soluble contrast myelography.[8] The two hypotheses advanced to account for the palsy are: (1) subsequent arachnoiditis which is the result of injection of the contrast material into the subarachnoid space; (2) caudal displacement of the brain due to CSF leakage, causing tension on the sixth nerve especially at the right angle bend.[8]

Management

The easiest management is occlusion of one eye to avoid diplopia since acquired sixth nerve palsy is often transient. However, this strategy is not appropriate for children since it may result in occlusion amblyopia and since it prevents binocularity during the critical period of visual development.

An alternative used in our case was Fresnel prism placed on the side with the suspected sixth nerve palsy. Half the amount of prism needed to neutralize the diplopia was prescribed to give the patient an opportunity to use fusional reserves. Anti-suppression and anaglyphic fusion training were prescribed in an effort to expand the patient's fusional range so that she would have a greater ability to compensate for her current diplopia. This method of management offers an advantage to monocular occlusion in that it preserves binocularity until such time that the palsy resolves.

Botulinum toxin injections also have been effective in managing acquired sixth nerve palsies. The antagonistic medial rectus muscle is weakened, decreasing or preventing medial rectus contracture. As the lateral rectus muscle begins to recover, alignment is facilitated by the relaxed medial rectus muscle.[9] Surgery could be eliminated or the amount of medial rectus recession reduced after botulinum toxin treatment.[9]

Management of sixth nerve palsy begins with a complete case history including history of trauma or cancer, medical health and associated symptoms with the palsy. A physical examination should be recommended. Ocular health, binocular vision and refraction must be examined.

Patients should be monitored regularly, and if diplopia persists or worsens within six months, such patients should be referred for a CT scan or MRI to rule out space-occupying lesions.

This case was referred by Dr P Nickolet, and was seen during the author's clinical externship at the Vision Institute of Canada. The author expresses her appreciation to the staff of the Institute for their assistance in the preparation of this case report.

References

1. Curran RE. True and simulated divergence palsy as a precursor of benign sixth nerve palsy. Binocular Vision Quarterly 1989:4;125–30.
2. Robertson DM, Hines JD, Rucker CW. Acquired sixth nerve paresis in children. Arch Ophthal 1970:83;547–79.
3. Shrader EC, Schlezinger NS. Neuro-ophthalmologic evaluation of abducens nerve paralysis. Arch Ophthal 1960:63;84–91.
4. Schneider RC, Johnson FD. Bilateral traumatic abducens palsy: A mechanism of injury suggested by the study of associated cervical spine fractures. J Neurosurg 1971:34;33–37.
5. Wolff EA. A bend in the sixth cranial nerve—and its probable significance. Brit J Ophthal 1828:12;22–24.
6. Sakalas R, Harbiseon JW, Vines FS, Becker DP. Chronic sixth nerve palsy: An initial sign of basisphenoid tumours. Arch Ophthal 1975:93;186–90.
7. Savino PJ, Hilliker JK, Cassell GH, Schatz NJ. Chronic sixth nerve palsies: Are they really harbingers of serious intracranial disease? Arch Ophthal 1982:100;1442–44.
8. Miller EA, Savino PJ, Schatz NJ. Bilateral sixth nerve palsy: A rare complication of water-soluble contrast myelography. Arch Ophthal 1982:100;603–4.
9. Metz HS, Dickey CF. Treatment of unilateral acute sixth nerve palsy with botulinum toxin. Am J Ophthal 1991:112;381–84.

Chemical soaking solutions versus hydrogen peroxide for soft lens disinfection: Is there a significant difference?

Desmond Fonn, Sheila Grunert

Twenty years have elapsed since the introduction of soft contact lenses, and in that period a host of disinfection solutions and systems have been introduced and re-introduced. Claims of efficacy, absence of toxicity and sensitivity, convenience, superiority and the like have not been uncommon in attempts to gain market share in what is an international billion-dollar business.

At present the two main disinfecting-system rivals are chemical soaking solutions and hydrogen peroxide. The other, less popular disinfection methods are the physical systems, which include thermal, ultrasound, ultra-violet, microwave and standing wave methods.

The issue of "which is better" will not be addressed in this paper as there is no simple or single answer, but the intention is to draw comparisons between chemical disinfection and hydrogen peroxide systems. Issues such as regulatory process of approval, efficacy, toxicity, convenience and compliance will be discussed.

Regulatory process of approval

The Health Protection Branch (HPB) of Health and Welfare Canada has, in the last two years, designated contact lens disinfecting systems as drugs. Prior to marketing any soft lens disinfecting system, application has to be made to HPB. Approval will likely be granted by this agency with the following support related to the product: proposed name for sale; quantitative lists of ingredients; name and address of formulator, compounder or packager; specifications of ingredients; packaging in contact with the product; drafts of labels, inserts and brochures; and specifications of dosage forms. The Food and Drugs Act and Regulations[1] requires that disinfecting agents obtain a Drug Identification Number (DIN) prior to sale of the products. In addition, evidence of safety, efficacy, toxicological testing and stability of the product has to be provided by the manufacturer, but no complete minimum standard for contact lens care products exists in Canada at present.

By contrast, in the United States contact lens products must be approved by the Food and Drug Administration (FDA) before they can be sold. The Ophthalmic Devices division of the FDA has published guidelines[2] for the procedures which have to be followed before submitting an application for solution or disinfecting system approval. The FDA does not conduct the tests and the onus is on the applicant to perform the tests and to submit the required information. The FDA employs numerous statisticians, microbiologists and outside clinical consultants to review the submitted information. Currently, the FDA requires results from three separate tests for demonstrating effectiveness of a disinfection system. They are the *contribution-of-elements* test, the *D-value* determination and the *multi-item microbial challenge* test.

The contribution-of-elements test involves each component's contribution to decreasing the microbial load. For example, the cleaning, disinfecting and rinsing steps are evaluated separately in the system's overall disinfecting capacity.

The second procedure examines the active disinfection process and as such is an out-of-regimen test. One million micro-organisms are added to one millilitre of the disinfection solution. The number of surviving micro-organisms is determined at specific times. The D-value, or decimal reduction time, is the time required for 90% inactivation of a population of organisms, that is, a one-log reduction calculated from the kill curve. The shorter the D-value, the more effective a given disinfectant is considered to be. D-values interpolate linear kill rates, which means that the time/reduction of micro-organisms is constant. This is not the case for all micro-organisms. While the D-values are sensitive to technique and different researchers have found widely different values, especially for fungal organisms,[3,4] the D-value test can be used to assess the relative capacity of a system to withstand a microbial challenge. The FDA requires that D-values be submitted for the same ocular pathogens as the multi-item microbial challenge test. The FDA uses the D-values to set the minimum disinfection soaking time for the system. This is set at three times the largest D-value.[3]

The multi-item microbial challenge test appears to be the most stringent in the battery of tests required by the FDA. It evaluates the entire disinfection regimen. The test uses both new and worn contact lenses (if possible), which are inoculated with a microbial preparation and an organic load or "soil" to mimic deposits on lenses from eye secretions. The challenge organisms are

D Fonn, DipOptom, MOptom, FAAO; S Grunert, OD—Centre for Contact Lens Research, School of Optometry, University of Waterloo

Staphylococcus epidermidis, Pseudomonas aeruginosa, Serratia marcescens, Candida albicans, Aspergillus fumigatus and *Herpes simplex*. Twenty test lenses from each of the four FDA-classified groups are inoculated with the solution containing one million of each of six different micro-organisms. This totals 480 lenses. After an absorption period the lenses are subjected to the complete disinfection system including cleaning, soaking and neutralizing or rinsing. For the system to be considered efficacious, there should be no viable micro-organisms on the lenses or in the solution remaining in the lens case.

There is one more issue concerning the approval process of chemical disinfecting solutions containing preservatives (antimicrobial agents), and that is the shelf life. The shelf-life sterility stability and the shelf-life stability of the preservative must be demonstrated by the manufacturer.

Efficacy of disinfecting systems

The fact that disinfecting systems have FDA or HPB approval suggests that they are effective when used according to manufacturers' directions. However the active ingredients of disinfecting systems are not equally effective, as judged by D-values when the disinfecting agent is tested on a stand-alone basis.[5–8]

Hydrogen peroxide (H_2O_2) is acknowledged as one of the best disinfectants for contact lenses,[9] but even this solution demonstrates the unequal resistance of micro-organisms. For example, some H_2O_2 systems are effective against *Pseudomonas aeruginosa* with a short exposure time (10 or 15 minutes) but ineffective against *Serratia marcescens* bacteria or *Candida* and *Aspergillus* fungi for the same exposure time.[4,10,11] Some questions surrounding D-values have arisen with respect to H_2O_2 because the results across different laboratories have not been repeatable. The table shows an example of the differing results. These differences may be credited to factors such as differences in experimental techniques, strains of organisms used, initial level of the microbial load and incubation conditions. D-values are based on the assumption of linear (constant) kill rates; because this may not be the case for many organisms that do not exhibit first-order death rate kinetics, it may therefore be an inappropriate method of reporting biocidal activity for some solutions.[3,12–15] However, D-values do serve a useful purpose in comparing the relative strengths of active disinfecting agents (out-of-regimen) from various systems.

Nonperoxide, large-molecule, chemical disinfecting systems are based on the principles of incorporating the lowest concentration of active ingredient that will produce disinfection with longer soak times but without causing toxicity. Another distinct advantage of these systems is their continuous antimicrobial activity. The D-values of current chemical systems such as Opti-Free and Sensitive Eyes Soaking Solution (ReNu in the United States), whose name has recently changed to Multi-Purpose Solution (MPS), are less impressive than H_2O_2 if the solutions are tested out-of-regimen. But the basis of these solutions is that they are part of a system and the cleaning and rinsing elements contribute significantly to the efficacy of the system.

Acanthamoeba is not a common ocular pathogen and is not included in the battery of tests for efficacy established by the FDA in the United States. However, *Acanthamoeba* infection has serious consequences and the risk of this ubiquitous organism to the eye of contact lens wearers should not be ignored. In evaluating the efficacy of a system against *Acanthamoeba*, the capacity for killing both cysts and trophozoites of at least the two most common types, *A. castellanii* and *A. polyphaga*, should be included.

Penley et al.[8] evaluated four soft contact lens disinfecting solutions, Softmate, ReNu (MPS), Hydrocare and Opti-Free and found that none of the disinfection solutions alone were effective against both cysts and trophs of both types. ReNu and Opti-Free were ineffective against cysts even at seven days. Davies et al.[7] tested five commercially available soft contact lens disinfectants; the results for ReNu, Opti-Soft and AOSept showed no measurable anti-*Acanthamoeba* activity. Hydrocare and Oxysept, on the other hand, were able to reduce the cyst challenge to zero in 4 to 24 hours. No trophozoite plaques were observable at the 24-hour time-point with either Hydrocare or Oxysept. The difference between Penley's and Davies' results of Hydrocare may be due to method or the initial inoculum.

To date, no reports have been published linking serious infections to either MPS or Opti-Free. In fact, three publications of clinical trials (six months to two

Table 1. Hydrogen peroxide D-values

Organism	Houlsby[18] (1984)	Janoff[16] (1984)	Sibley[17] (1983)	Penley[4] (1985)	Reinhardt[5] (1990)
S. aureus	–	–	2.35	–	<20
S. epidermidis	1.7	1.3	1.62	–	<20
P. aeruginosa	1.7	5.3	0.40	–	<20
S. marcescens	3.1	3.7	–	2.8	<20
A. fumigatus	25.1	6.5	–	11	<20
C. albicans	27.9	10.3	–	48	–

years) with these products did not report any clinically significant reactions among the 700 patients in these clinical trials.[19–21] However, Roth[22] reported Polyquad implicated adverse responses in eight patients, but acknowledged that the reactions were probably due to the combination of other solutions with Polyquad.

Continuous antimicrobial activity

Patients who have more than one set of lenses or those who only wear their lenses occasionally will obviously benefit from products that provide continuous antimicrobial activity. Unfortunately this is not born out in practice. Campbell and Caroline[23] found soft lens disinfection techniques were generally ineffective in the home environment. Twenty-nine per cent of the heat, 50% of the H_2O_2, and 75% of the chemical solution disinfected cases cultured positive for microbial growth. These patients supposedly performed correct disinfection procedures. Similar findings were recorded by Pitts and Krachmer.[24] Caroline and Campbell[25] have speculated that the biofilm that forms in the case is secreted by the bacterial cells, which protects the bacteria from inactivation. Additionally, laboratory-cultured bacteria and "street" bacteria may be of different strains and have different resistance capacities.

Fortunately, the incidence of clinically significant microbial keratitis is surprisingly low. One study found an incidence of 0.04% of microbial ocular infections among daily soft lens wearers.[26] The determination of why the incidence of infection is low has yet to be established in view of the fact that patient storage cases and solutions have been found to be contaminated. It appears that bacteria entrapped in a biofilm are protected from antimicrobial agents or surfactants.[27] Simmons et al.[28] sampled cases from both H_2O_2 and chemical (non-peroxide) users and found that both groups' cases (19%) were contaminated, while Wilson et al.[29] found that H_2O_2 was more effective against biofilms than solutions formulated with chlorhexidine gluconate, polyquarternium -1 or polyaminopropyl biguanide (Dymed). Polyaminopropyl biguanide has been found to be effective against biofilm, but this is only available in the Boston Advance rigid conditioning solution at a concentration of 0.0015%, 30 times higher than the Dymed concentration in MPS. It appears that more effective soft lens disinfection products that can penetrate and neutralize the biofilm have to be developed. As an alternative, cases that can be easily and frequently cleaned or that are disposable should be used.

Toxicity and ocular reactions

Ocular exposure to 3% hydrogen peroxide has adverse effects. The acute reaction includes intense pain, hyperemia, edema, excessive corneal and conjunctival epithelial cell loss and a transient reduction in vision. This may occur if patients forget the all-important neutralization procedure of two-step H_2O_2 systems. Neutralization is supposed to reduce the peroxide concentration to zero parts per million (ppm), or a subthreshold level, restore the pH of the solution to approximately 7.3, restore the osmolarity to an equivalent of 0.9% and restore the lens parameters to pre-disinfection state.

Transient low-grade stinging upon insertion is a more common complaint. This is one of the reasons that patients may avoid disinfecting their lenses when using a hydrogen peroxide system. The two probable causes of stinging are residual H_2O_2 in the lens after neutralization and/or final pH of the system being outside the normal range. Estimates of the H_2O_2 threshold level that results in clinical symptoms of irritation range from less than 10 ppm to over 250 ppm.[16,30–32] Chalmers and McNally[32] studied ocular detection thresholds of H_2O_2 drops instilled directly into the eye as well as lenses soaked in the same solutions. Their results of threshold discomfort levels ranged from 490 to 1,470 ppm for drops and 210 to 408 ppm for soaked lenses, and their conclusion was that ocular detection thresholds may be significantly higher than previously stated.

Chalmers et al.[33] reported on H_2O_2 removal from 74% water content lenses while in the eye. They found that at least 90% of the H_2O_2 was removed within the first 30 seconds and complete removal was achieved within 60 seconds. The closed eye showed a faster rate of removal than the open blinking eye, which led them to the conclusion that active neutralization from ocular catalase, rather than dilution of drainage down the nasolacrimal duct, is responsible for the rapid H_2O_2 removal from the lens. Although the residual H_2O_2 levels of various systems have been shown to differ,[34–38] at least one study[39] has demonstrated that patient comfort ratings of these H_2O_2 systems (post-neutralization) are not statistically different and all produced very little to no stinging on lens insertion. Riley and Kast[40] reported that neither corneal endothelium nor other intraocular tissues will be damaged by residual concentrations of H_2O_2 up to 680 ppm from contact lens wear with intact epithelium.

It has been proposed that patients who disinfect their soft lenses with H_2O_2 and report discomfort on insertion may do so as a result of an acidic shift of the pH. The ocular awareness pH threshold is reported to be outside the range of 6.6 to 7.8. Harris,[35] however, found that after neutralization only two systems (Lensept and generic H_2O_2) had residual pH values beyond this range.

Another element of concern related to the toxicity of H_2O_2 is its long-term effect. This is a highly controversial topic. Some investigators have found that increased levels of H_2O_2 (i.e., beyond the 26 ppm that is normally present in the aqueous humor) may induce cataracts[41] and cause corneal endothelial dysfunction,[42] contrary to what Riley and Kast reported.[40] An in-vitro study demonstrated

epithelial cell damage when exposed to H_2O_2 at concentrations between 30 and 100 ppm.[43]

Hydrogen peroxide became popular as a result of thermal disinfection's accelerated denaturation of lens protein build-up and its adverse effect on medium- and high-water-content contact lenses as well as adverse ocular responses to preservatives in chemical disinfecting solutions. The two preservatives that were responsible for delayed hypersensitivity or allergic reactions with soft lens use were chlorhexidine gluconate and thimerosal.[44–46] Two of the possible reasons for the ocular reactions to these two antimicrobial agents were that concentrations might have been too high and the molecular sizes of the preservatives were smaller than the pore size of hydrogel materials (chlorhexidine 32 angstroms and thimerosal 13 angstroms).

Two of the current chemical disinfecting solutions which use preservatives are MPS, which contains Dymed (the proprietary name) in a concentration of 0.00005%, and Opti-Free, which contains Polyquad at 0.001% as the antimicrobial agent. Both the Dymed and Polyquad molecule sizes, at 144 and 225 angstroms respectively, are greater than the hema pore size of 30 to 50 angstroms and thus do not penetrate the lens matrix.

In the four years that these two products have been on the market, no reports of adverse ocular responses have emerged (except for the Roth paper[22]) in the form of hypersensitivity. In fact a number of large clinical trials have been reported[19–21] showing very favourable results: i.e., an absence of subjective symptoms and insignificant ocular signs.

Convenience and compliance

Between 40 and 74% of contact lens wearers are noncompliant in caring for their contact lenses according to reports in the literature.[47–49] Patients may be guilty of noncompliance by using insufficient solution, repeatedly re-using the same solution and not cleaning out the container, using solutions beyond the expiry date, skipping the precleaning step, skipping the disinfection step or not disinfecting for the required period and failing to replace the catalytic disk at the stipulated intervals. These common errors and omissions may increase the risk of contamination and infection. The fact that so many patients' cases, contact lens care systems and lenses have been found to be microbially contaminated[23,50–52] would implicate their compliance, and at least one author[53] has suggested that noncompliance can result in microbial infection.

The contributions of the cleaning, disinfecting and rinsing steps ensure the efficacy and safety of a system. This has been elaborated on by Efron et al.,[54] who implicated the omission of surfactant cleaning and rub-and-rinse in patients who developed *Pseudomonas* corneal ulcers with daily wear disposable lenses.

There are many reasons why patients are noncompliant: inadequate patient education, complacency, expense of solutions, some products causing ocular irritation, complexity of some systems and the abundance of different brands of products available when replacements are required. Compliance may be enhanced by improving patient education, recommending products specific to patients' wearing needs and using simpler or more convenient systems (fewer products for cleaning, disinfection and rinsing). This has been demonstrated in other disciplines of medicine where simpler care regimens appear to have increased compliance.[55]

It would seem that chemical systems are being prescribed more frequently than H_2O_2 in the United States.[56] In Canada, the percentages are much closer, as opposed to a few years ago where H_2O_2 was the much more popular system. Studies of patients' preferences when asked to compare ReNu, Opti-Free and AOSept[57–58] resulted in MPS being preferred for convenience (ease of use) in both studies but AOSept over the other two for comfort in the Steel study.[58]

Conclusion

This paper has emphasized that currently available and FDA-approved H_2O_2 and chemical (nonperoxide) disinfection systems should be safe and disinfect lenses equally well if the entire cleaning, disinfection and rinsing procedure of each system is strictly followed. The FDA requirements appear to be stringent enough for safety and efficacy even though organisms such as *Acanthamoeba* are not included in the battery of tests.

Hypersensitivity reactions to H_2O_2 and new-generation chemical systems have not been reported other than acute toxic reaction to H_2O_2 if lenses have not been neutralized. One would assume that if the nonperoxide systems cause hypersensitivity, some reports would have appeared by this stage, as the solutions have been used in many countries for at least four years. Convenience, simplicity or both may be important factors in improving compliance, and, according to Schwartz's survey,[56] compliance appears to be increasing.

The authors would like to thank the following people for their valuable comments: Drs Murchison Callender, Keith Gordon, Martin McDowell, Bill Reindel, Luigina Sorbara and David Wintermeyer; Ms Lorraine Johnson; and Ms Brenda Minno.

References

1. Health Protection Branch, Health and Welfare Canada. Information Letter #774, February 20, 1990.
2. Testing Guidelines for class III soft (hydrophilic) contact lens solutions. Center for Devices and Radiological Health, Dept of Health and Human Services, United States Food and Drug Administration 1985;44–55.

3. Janoff LE. Origin and development of hydrogen peroxide disinfection systems. CLAO J 1990;16:S30–42.
4. Penley CA, Llabrés C, Wilson LA, Ahearn DG. Efficacy of hydrogen peroxide disinfection systems for soft contact lenses contaminated with fungi. CLAO J 1985;11:65–8.
5. Reinhardt DJ, Kaylor B, Presscott D, Sapps CS. Rapid and simplified comparative evaluations of contact lens disinfecting solutions. ICLC 1990;17–9–12.
6. Anger CB, Ambrus K, Stoeker J, et al. Antimicrobial efficacy of hydrogen peroxide for contact lens disinfection. Contact Lens Spectrum 1990;5(11):46–51.
7. Davies DJG, Anthony Y, Meakin BJ, et al. Evaluation of the antiacanthamoeba activity of five contact lens disinfectants. ICLC 1990;17:14–20.
8. Penley CA, Willis SW, Sickler SG. Comparative antimicrobial efficacy of soft and rigid gas permeable contact lens solutions against acanthamoeba. CLAO J 1989;15:257–60.
9. Holden BA. Report card on hydrogen peroxide for contact lens disinfection. CLAO J 1990;16:S61–64.
10. Levine WL, Litsky W, Lamm RA. Disinfection of hydrophilic contact lenses with commercial preparations of 3% and 6% hydrogen peroxide. Dev Ind Microbiol 1981;22:813–19.
11. Campbell JE, Dimick RL. Effect of 3% hydrogen peroxide on the viability of Serratia marcescens. J Bacteriol 1966;91:925–29.
12. Lowe R, Brennan NA. Hydrogen peroxide disinfection of hydrogel contact lenses: an overview. Clin Exp Optom 1987;70:190–97.
13. Mowrey-McKee MF, Busschaert SC, Hamberger J, et al. A comparison of bacterial death rate kinetics among contact lens solutions. Presented at 89th meeting of the American Society for Microbiology, New Orleans, LA, May 1989.
14. Sutton SVW, Franco RJ, Porter DA, et al. D-value determinations are an inappropriate measure of disinfecting activity of common contact lens disinfecting solutions. Appl Environ Microbiol 1991;57:2021–26.
15. Lowe R, Vallas V, Brennan NA. Comparative efficacy of contact lens disinfecting solutions. CLAO J 1992;18:34–40.
16. Janoff LE. The Septicon™ system: a review of pertinent scientific data. ICLC 1984;11:274–79.
17. Sibley M. Hudrogen peroxide care systems. Contact Lens Forum 1987;12(7):57–63.
18. Houlsby R, Ghajar M, Chavez G. Microbiological evaluation of soft contact lens disinfecting solutions. J Am Optom Assoc 1984;55:205–11.
19. Reindel WT, Huber JM, Proskin HM. Multipurpose products—The patient's preference. Contact Lens Forum 1989;14(9):67–72.
20. Leisring J, Gill L. The clinical safety of a new generation chemical disinfecting agent. Contact Lens Spectrum 1990;5(10):63–67.
21. Schachet J, Lowther GE, Lavaux JE, et al. Clinical evaluation of the Opti-Free disinfection system. Contact Lens Spectrum 1990;5(12):37–41.
22. Roth HW. Polyquad induced mixed solution syndrome in contact lens wear. Contactologia 1991;13;8–11.
23. Campbell RC, Caroline PJ. Inefficacy of soft contact lens disinfection techniques in the home environment. Contact Lens Forum 1990;15(9):17–26.
24. Pitts RE, Krachmer JH. Evaluation of soft contact lenses in the home environment. Arch Ophthalmol 1979;97:470–72.
25. Caroline PJ, Campbell RC. Strategies of microbial cell survival in contact lens cases. Contact Lens Forum 1990;15(10):27–36.
26. Poggio EC, Glynn RJ, Schein OD, et al. The incidence of ulcerative keratitis among users of daily-wear and extended-wear soft contact lenses. N Engl J Med 1989;321(12):779–83.
27. Chou MH, DeCiccio BT, Keevan JK, et al. The mechanism of survival of bacteria in contact lenses cases. Presented at the Contact Lens Association of Ophthalmologists meeting, New Orleans, LA, January 1989.
28. Simmons PA, Edrington TB, Hsieh L, Wang LW. The incidence of bacterial contamination in contact lens case caps and wells: A comparison among cold chemical disinfection systems. Optom Vis Sci 1990;67(supp):70.
29. Wilson LA, Sawant Ad, Ahearn DG. Comparative efficacies of soft contact lens disinfectant solutions against microbial films in lens cases. Arch Ophthalmol 1991;108:1155–57.
30. Janoff LE. The effective disinfection of soft contact lenses using hydrogen peroxide. Contacto 1979;23:37–40.
31. Paugh JR, Brennan NA, Efron N. Ocular response to hydrogen peroxide. Am J Optom Physiol Opt 1988;65:91–98.
32. Chalmers RL, McNally JJ. Ocular detection threshold for hydrogen peroxide: drops vs. lenses. ICLC 1988;15:351–57.
33. Chalmers RL, Tsao M, Scott G, Roth L. The rate of in vivo neutralisation of residual H_2O_2 from hydrogel lenses. Contact Lens Spectrum 1989;4(7):21–26.
34. Kelly W, Ward G, Williams W, Dziabo A. Eliminating hydrogen peroxide residuals in solutions and contact lenses. Contact Lens Spectrum 1990;5(1):41–44.
35. Harris MG. Practical considerations in the use of hydrogen peroxide disinfection systems. CLAO J 1990;16:S53–60.
36. Krezanoski JZ, Houlsby RD. A comparison of new hydrogen peroxide disinfection systems. J Am Optom Assoc 1988;59:193–97.
37. Gyulai P, Dziabo A, Kelly W, et al. Relative neutralisation ability of six hydrogen peroxide disinfection systems. Contact Lens Spectrum 1987;2(5):61–68.
38. Sibley MJ. Hydrogen peroxide residues. Contact Lens Spectrum 1988;3(8):39–43.
39. Melton JW, Phillips JH. Patient comfort comparison of hydrogen peroxide systems. Contact Lens Spectrum 1988;3(9):48–51.
40. Riley MV, Kast M. Penetration of hydrogen peroxide from contact lenses or tear-side solutions into the aqueous humor. Optom Vis Sci 1991;68:546–51.
41. Spector A. Garner WH. Hydrogen peroxide and human cataract. Exp Eye Res 1981;33:673–81.
42. Hull DS, Csukas S, Green K, Livingston V. Hudrogen peroxide and corneal endothelium. Acta Ophthalmol 1981;59:409–21.
43. Tripathi BJ, Tripathi RC. Hydrogen peroxide damage to human corneal epithelial cells in vitro. Arch Ophthalmol 1989;107:1516–19.
44. Peterson NB. Allergic contact conjunctivitis from merthiolate in soft contact lenses. Contact Dermatitis 1978;4:165.
45. Shaw EL. Allergies induced by contact lens solution. Contact Intraocular Lens Med J 1980;6:273–77.
46. Josephson J, Caffery B. Hydrogel lens solutions. International Ophthalmol Clin 1981;21(2):163–71.
47. Sokol JL, Mier MG, Bloom S, Asbell PA. A study of patient compliance in a contact lens wearing population. CLAO J 1990;16:209–13.
48. Chun MW, Weissman BA. Compliance in contact lens care. Am J Optom Physiol Opt 1987;64:274–76.
49. Collins MJ, Carney LG. Patient compliance and its influence on contact lens wearing problems. Am J Optom Physiol Opt 1986;63:952–56.
50. Donzis PB, Mondino BJ, Weissman B, Bruckner DA. Microbial contamination of contact lens care systems. Am J Ophthalmol 1987;104:325–33.
51. Donzis PB, Mondino BJ, Weissman BA, Bruckner DA. Microbial analysis of contact lens care systems contaminated with Acanthamoeba. Am J Ophthalmol 1989;108:53–56.
52. Wilson LA, Sawant AD, Simmons RB, Ahearn DG. Microbial

contamination of contact lens storage cases and solutions. Am J Ophthalmol 1990;110:193–98.
53. Moore MB. Acanthamoeba keratitis and contact lens wear: the patient is at fault. Cornea 1990;9:S33–35.
54. Efron N, Wohl A, Toma NG, et al. Pseudomonas corneal ulcers associated with daily wear of disposable hydrogel contact lenses. ICLC 1991;18:46–52.
55. Eisen SA, Miller DK, Woodward RS, et al. The effect of prescribed daily dose frequency on patient medication compliance. Arch Intern Med 1990;150:1881–84.
56. Schwartz CA. Drive to simplify systems, ensure efficacy marks developments. Contact Lens Forum 1989: 14(9):21–32.
57. Hammersla K, Coviello D. The consumer challenge: three care systems compared. Contact Lens Spectrum 1991;6(6):21–27.
58. Steel S. Patient preference study compares top lens care systems. Contact Lens Spectrum 1990;5(10):56–59.

Annual index 1992/93 Index annuel 1992/93

Each title is followed by a code that designates the category. The categories used in this volume are listed below.

Chaque titre est suivi d'un code qui indique dans quelle catégorie appartient. Les catégories utilisées dans ce volume sont les suivantes.

(A): Article
(AR • RA): Annual Report • Rapport annuel
(CC): Clinical Comment
(CD): Clinical Diagnosis
(CR): Case Reports
(E): Editorial • Éditorial
(F): Feature
(L): Letter
(PP • MP): President's Podium • Mot du président
(VCN): Vision Care News

- A -

accommodation
Précision de la réponse accommodative à un changement de vergence de la lumière (Kergoat and Lovasik) (A), 68

acuité visuelle
Mesure de l'acuité visuelle par la technique du regard préférentiel : Revue des méthodes (Laurin and others) (A), 12

adenoma
A tale of two fields (Williams) (CR), 147

aged
Study targets seniors and mobility (VCN), 39

Allergan
Allergan Pathways program (VCN), 98

allergic reactions
Ocular symptoms of allergies (VCN), 163

American Academy of Optometry
New AAO inductees (VCN), 38

American Optometric Association (AOA)
After a tough day at the office ... (VCN), 161
AOA Distinguished Service Award (VCN), 102
Dr Jean-Marie Rodrigue addresses AOA (VCN), 158

American Optometric Foundation
Canadian wins AOF fellowship (VCN), 220

Annual Report, CAO, 83

aphakia, unilateral
A trio of case reports (Chiarelli) (CR), 18

Article
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle), 132
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle), 180
Hockey faceguards and visual field scotomas (Wingert and others), 143
How useful is the VisTech MCT8000 for contrast sensitivity and glare testing? (Elliott), 194
Mesure de l'acuité visuelle par la technique du regard préférentiel : Revue des méthodes (Laurin and others), 12
Précision de la réponse accommodative à un changement de vergence de la lumière (Kergoat and Lovasik), 68
La sensibilité au contraste : une réalité clinique. I. Mesure et interpretation (Bolduc and others), 62
La sensibilité au contraste : une réalité clinique. II. Implications cliniques (Bolduc and Gresset), 126

Asian Pacific Optometric Congress
Call for papers--APOC 1993 (VCN), 41

l'Association canadienne des optométristes (ACO)
Groupe de travail national sur la Charte constitutive de l'ACO (RA), 88
Rapport annuel, 83

astigmatism
Symposium Report: Building a practice with soft toric contact lenses (Gerber) (F), 30

Atlantic Optometric Continuing Education Symposium
Atlantic CE program set (VCN), 38

awards
CAO 1991 awards (AR), 85
Fight for Sight awards (VCN), 222
Indian MD wins humanitarian award (VCN), 222
Lighthouse calls for 1993 humanitarian nominations (VCN), 222
Waterloo 1992 news (VCN), 158

- B -

Bausch & Lomb
B&L donate to Vision Institute (VCN), 100
B&L launches environmentally friendly packaging (VCN), 224
B&L Olympic screening success (VCN), 105
B&L's research symposia merge (VCN), 224
Canadians win poster contest (VCN), 224
IACLE receives strong commitment of industrial support (VCN), 40
New technical consultation line (VCN), 224
Stats from B&L's Olympic screening (VCN), 224

Beaulne C, see **Laurin L** Jt Auth

Bolduc M, Gresset J, La sensibilité au contraste : une réalité clinique. II. Implications cliniques (A), 126
Bolduc M, Simonet P, Gresset J, La sensibilité au contraste : une réalité clinique. I. Mesure et interpretation (A), 62
Braun M, Lyle WM
Corneal dystrophies and degenerations. Part 1: The anterior layers (A), 132
Corneal dystrophies and degenerations. Part 2: The stroma (A), 180
Breker JA, Eye care professionals needed in Ukraine (L), 120

- C -

Canada Winter Games
PEI members active at 1991 Winter Games (VCN), 97
Canadian Association of Optometrists (CAO)
Annual report, 83
National Task Force on the Constitution (AR), 87
Canadian Optometric Education Trust Fund (COETF), Annual report, 88
cartes d'acuité
Mesure de l'acuité visuelle par la technique du regard préférentiel : Revue des méthodes (Laurin and others) (A), 12
cartes de crédit
Carte d'affinité or (RA), 87
Case Reports
Acquired sixth nerve palsy (Marconi), 200
A tale of two fields (Williams), 147
A trio of case reports (Chiarelli), 18
Unusual presentations of retinal detachment (Williams), 73
cataract, congenital
A trio of case reports (Chiarelli) (CR), 18
cerebral palsy
A trio of case reports (Chiarelli) (CR), 18
Chiarelli C
Doyne's honeycomb dystrophy (CD), 177
Morning glory disc (CD), 60
Non-arteritic anterior ischemic optic neuropathy (CD), 123
A trio of case reports (CR), 18
child
Which way is the blackbird flying? (VCN), 163
Christian Blind Mission International
Essilor provides boost to CBMI (VCN), 222
CIBA Vision
CIBA introduces new saline (VCN), 224
CIBA invests in Japan (VCN), 224
Clinical Comment
Chemical soaking solutions versus hydrogen peroxide for soft lens disinfection (Fonn and Grunert), 202
Position paper on disposable contact lenses (International Committee on Contact Lenses), 79
Clinical Diagnosis
Doyne's honeycomb dystrophy (Chiarelli), 177
Morning glory disc (Chiarelli), 60
Non-arteritic anterior ischemic optic neuropathy (Chiarelli), 123
clinical signs
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle) (A), 132
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle) (A), 180
COETF, see **Canadian Optometric Education Trust Fund (COETF)**
contact lenses
"Accelerated" Ortho-K announced (VCN), 163
contact lenses, disposable
Position paper on disposable contact lenses (International Committee on Contact Lenses) (CC), 79
contact lenses, soft toric
Symposium Report: Building a practice with soft toric contact lenses (Gerber) (F), 30
contact lens solutions
Chemical soaking solutions versus hydrogen peroxide for soft lens disinfection (Fonn and Grunert) (CC), 202
Federal issues 1991 (AR), 83
contrast sensitivity
How useful is the VisTech MCT8000 for contrast sensitivity and glare testing? (Elliott) (A), 194
corneal dystrophy
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle) (A), 132
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle) (A), 180
credit cards
Affinity gold card (AR), 87

- D -

Décarie R
Borrowing to invest (FF), 218
Emprunter pour investir (PF), 215
Le fisc et votre revenu de placement (PF), 33
Foreign assets (FF), 216
How your invest income is taxed (FF), 35

Pay down your mortgage or make an RSP contribution? (FF), 95
Les régimes du conjoint (PF), 34
Remboursez votre prêt hypothécaire tout en investissant dans votre RÉR (PF), 94
Le RÉR autogéré (PF), 149
Self-directed RSPs (FF), 155
Spousal plans (FF), 36
Les titres étrangers (PF), 214
degenerations
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle) (A), 132
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle) (A), 180
developing countries
Operation Eyesight (VCN), 222
diagnosis, differential
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle) (A), 132
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle) (A), 180
Unusual presentations of retinal detachment (Williams) (CR), 73
diplopia
Acquired sixth nerve palsy (Marconi) (CR), 200
disability glare
How useful is the VisTech MCT8000 for contrast sensitivity and glare testing? (Elliott) (A), 194
disinfection systems
Chemical soaking solutions versus hydrogen peroxide for soft lens disinfection (Fonn and Grunert) (CC), 202
dystrophies, of the anterior layers
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle) (A), 132
dystrophies, Doyne's honeycomb (Chiarelli) (CD), 177
dystrophies, of the stroma
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle) (A), 180

- E -

Editorial
Boundary disputes and ulterior motives? (Spear), 4
The lure (Samek), 115
Once again! (Samek), 172
Prudent management (Reid), 52
Reciprocity--a right! (Hart), 4
Responsibility! (Samek), 50
Éditorial
L'attrait (Samek), 115
Encore une fois! (Samek), 172
Prudence dans le traitement (Reid), 52
La responsabilité (Samek), 50
Elliott DB, How useful is the VisTech MCT8000 for contrast sensitivity and glare testing? (A), 194
equipment and supplies
Collecting ophthalmic antiques (VCN), 162
File this under "nice try" (VCN), 164
How useful is the VisTech MCT8000 for contrast sensitivity and glare testing? (Elliott) (A), 194
Lighthouse shines on early vision needs (VCN), 105
Low vision aids for keyboards (VCN), 107
New field analyzer (VCN), 105
New technology for eye topography (VCN), 40
Ocutech receives Ontario grant (VCN), 161
Pinhole glasses' curative claims shot full of holes (VCN), 100
Poster illustrates common vision problems (VCN), 105
Recording dynamic visual acuity (VCN), 107
Space in your waiting room library? (VCN), 107
Which way is the blackbird flying? (VCN), 163
Essilor Canada
Essilor provides boost to CBMI (VCN), 222
Montreal School and Essilor reach agreement (VCN), 97
eyeglasses
Pinhole glasses' curative claims shot full of holes (VCN), 100

- F -

Financial Foresight
Borrowing to invest (Décarie), 218
Foreign assets (Décarie), 216
How your invest income is taxed (Décarie), 35
Pay down your mortgage or make an RSP contribution? (Décarie), 95
Self-directed RSPs (Décarie), 155
Spousal plans (Décarie), 36
Le Fonds de fiducie des optométristes canadiens pour l'éducation
Rapport annuel, 89
Fonn D, Grunert S, Chemical soaking solutions versus hydrogen peroxide for soft lens disinfection (CC), 202

fréquences spatiales
La sensibilité au contraste : une réalité clinique. I. Mesure et interpretation (Bolduc and others) (A), 62
La sensibilité au contraste : une réalité clinique. II. Implications cliniques (Bolduc and Gresset) (A), 126

- G -

Gerber P, Symposium Report: Building a practice with soft toric contact lenses: Canadian panellists cite some simple steps to success (F), 30
Giasson C, see **Lapierre M** Jt Auth
Gresset J, see **Bolduc M** Jt Auth; **Laurin L** Jt Auth
Grunert S, see **Fonn FD** Jt Auth

- H -

Hansen des Groseilliers MR
A new responsibility for Canadian Optometry (PP), 117
Une nouvelle responsabilité de l'optométrie canadienne (MP), 117
L'optométrie célèbre ses 25 ans à Waterloo (MP), 175
Optometry's celebration of 25 years at Waterloo (PP), 175
Hart LG, Reciprocity--a right! (E), 4
head protective devices
Hockey faceguards and visual field scotomas (Wingert and others) (A), 143
health law
Law firm publishes health bulletin (VCN), 222
histological features
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle) (A), 132
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle) (A), 180
history
Collecting ophthalmic antiques (VCN), 162
hydrogen peroxide
Chemical soaking solutions versus hydrogen peroxide for soft lens disinfection (Fonn and Grunert) (CC), 202

- I -

impact fonctionnel
La sensibilité au contraste : une réalité clinique. II. Implications cliniques (Bolduc and Gresset) (A), 126
implications cliniques
La sensibilité au contraste : une réalité clinique. II. Implications cliniques (Bolduc and Gresset) (A), 126
inheritance pattern
Corneal dystrophies and degenerations. Part 1: The anterior layers (Braun and Lyle) (A), 132
Corneal dystrophies and degenerations. Part 2: The stroma (Braun and Lyle) (A), 180
International Association of Contact Lens Educators
IACLE receives strong commitment of industrial support (VCN), 40
International Committee on Contact Lenses,
Position paper on disposable contact lenses (CC), 79
International Optometric and Optical League (IOOL)
Optometrist of the Year (VCN), 102
IOOL, see **International Optometric and Optical League (IOOL)**

- K -

Kergoat H, Lovasik JV, Précision de la réponse accommodative à un changement de vergence de la lumière (A), 68

- L -

Laurin L, Gresset J, Beaulne C, Mesure de l'acuité visuelle par la technique du regard préférentiel : Revue des méthodes (A), 12
learning disability
A trio of case reports (Chiarelli) (CR), 18
Letter
Dispensing with smokescreens (Slaght), 59
Eye care professionals needed in Ukraine (Breker), 120
Iridology show sparks response (Wood), 118
Long WF, see **Wingert TA** Jt Auth
Lovasik JV, see **Kergoat H** Jt Auth
lupus
Lupus explained (VCN), 164
Lyle WM, see **Braun M** Jt Auth

- M -

Manitoba Association of Optometrists (MAO)
Manitoba ODs "unsociable" (VCN), 226
Manitoba Optometric Society (MOS), see **Manitoba Association of Optometrists (MAO)**
Marconi M, Acquired sixth nerve palsy (CR), 200

Marfan's syndrome
Ocular signs of Marfan's syndrome (VCN), 39
Martin WG, see **Wingert TA** Jt Auth
morning glory disc (Chiarelli) (CD), 60
Mot du président
Assurance-maladie au Canada (Rodrigue), 8
Une nouvelle responsabilité de l'optométrie canadienne (Hansen des Groseilliers), 117
L'optométrie célèbre ses 25 ans à Waterloo (Hansen des Groseilliers), 175
Regards sur le passe et l'avenir (Rodrigue), 53

- N -

New Brunswick Association of Optometrists
Atlantic action (VCN), 42
Newfoundland Association of Optometrists
Atlantic action (VCN), 42
Atlantic CE program set (VCN), 38
non-arteritic anterior ischemic optic neuropathy (Chiarelli) (CD), 123
nourrissons
Mesure de l'acuité visuelle par la technique du regard préférentiel : Revue des méthodes (Laurin and others) (A), 12
Nova Scotia Association of Optometrists
Atlantic action (VCN), 42

- O -

Olympic Games
B&L Olympic screening success (VCN), 105
Stats from B&L's Olympic screening (VCN), 224
Optometric Extension Program Foundation
OEP catalogue available (VCN), 104

- P -

palsy
Acquired sixth nerve palsy (Marconi) (CR), 200
A trio of case reports (Chiarelli) (CR), 18
paresis, superior oblique
A trio of case reports (Chiarelli) (CR), 18
passeports
Lettre de l'honorable Barbara McDougall, 57
Questions fédérales en 1991 (RA), 83
passports
Federal issues 1991 (AR), 83
Letter from the Honourable Barbara McDougall, 57
PEI Association of Optometrists
Atlantic action (VCN), 42
People
Baldwin, William, 102
Basman, Harry, 88
Giasson, Claude, 220
Gimbel, Howard, 220
Layland, Brian, 102
Lidiard, Victoria, 220
Rodrigue, Jean-Marie, 158
Venkataswamy, G, 222
Wall, Chantal, 41
Young-Carrière, Pamela, 104
Personnes
Basman, Harry, 88
President's Podium
Looking back, looking ahead (Rodrigue), 53
Medicare in Canada (Rodrigue), 8
A new responsibility for Canadian Optometry (Hansen des Groseilliers), 117
Optometry's celebration of 25 years at Waterloo (Hansen des Groseilliers), 175
Prévisions financières
Emprunter pour investir (Décarie), 215
Le fisc et votre revenu de placement (Décarie), 33
Les régimes du conjoint (Décarie), 34
Remboursez votre prêt hypothécaire tout en investissant dans votre RÉR (Décarie), 94
Le RÉR autogéré (Décarie), 149
Les titres étrangers (Décarie), 214
prix
Prix d l'ACO 1991 (RA), 86

- R -

Rapport Annuel, ACO, 83
regard préférentiel
Mesure de l'acuité visuelle par la technique du regard préférentiel : Revue des méthodes (Laurin and others) (A), 12
Reid A
Prudence dans le traitement (E), 52
Prudent management (E), 52
repeatability
How useful is the VisTech MCT8000 for contrast sensitivity and glare testing? (Elliott) (A), 194
retinal detachment
A tale of two fields (Williams) (CR), 147
Unusual presentations of retinal detachment (Williams) (CR), 73
Rodrigue J-M
Assurance-maladie au Canada (MP), 8
Looking back, looking ahead (PP), 53
Medicare in Canada (PP), 8
Regards sur le passe et l'avenir (MP), 53

- S -

Samek MJ
L'attrait (E), 115
Encore une fois! (E), 172
The lure (E), 115
Once again! (E), 172
La responsabilité (E), 50
Responsibility! (E), 50
scotoma
Hockey faceguards and visual field scotomas (Wingert and others) (A), 143
A tale of two fields (Williams) (CR), 147
Semaine de la vision (RA), 86
sensibilité au contraste
La sensibilité au contraste : une réalité clinique. I. Mesure et interpretation (Bolduc and others) (A), 62
La sensibilité au contraste : une réalité clinique. II. Implications cliniques (Bolduc and Gresset) (A), 126
seuil de contraste
La sensibilité au contraste : une réalité clinique. I. Mesure et interpretation (Bolduc and others) (A), 62
Simonet P, see **Bolduc M** Jt Auth
Simon Fraser University
New women's science centre (VCN), 104
Slaght RP, Dispensing with smokescreens (L), 59
Society of Canadian Women in Science and Technology
New women's science centre (VCN), 104
soins de santé aux tiers
Un programme national de soins de la vue aux tiers (RA), 85
solutions désinfectantes pour lentilles cornéennes
Questions fédérales en 1991 (RA), 83
Spear P, Boundary disputes and ulterior motives? (E), 4
sports
Hockey faceguards and visual field scotomas (Wingert and others) (A), 143
See also **Canada Winter Games**; **Olympic Games**
Steben M, see **Lapierre M** Jt Auth

- T -

techniques de mesure
La sensibilité au contraste : une réalité clinique. I. Mesure et interpretation (Bolduc and others) (A), 62
third-party health care
A national third-party program (AR), 84

- U -

United States National Society to Prevent Blindness
Fight for Sight awards (VCN), 222
Université de Montréal, École d'optométrie
Allergan Pathways program (VCN), 98
Montreal faculty translates US guide (VCN), 160
Montreal School and Essilor reach agreement (VCN), 97
President-elect addresses Montreal convocation (VCN), 38
University of Toronto, Faculty of Medicine
U of T health publication (VCN), 40
University of Waterloo, School of Optometry
Allergan Pathways program (VCN), 98
Waterloo 1992 news (VCN), 158
Waterloo CE program (VCN), 38

- V -

vergence de la lumière
Précision de la réponse accommodative à un changement de vergence de la lumière (Kergoat and Lovasik) (A), 68
Vision Awareness Week (AR), 86
vision, couleur
Précision de la réponse accommodative à un changement de vergence de la lumière (Kergoat and Lovasik) (A), 68
Vision Institute, Toronto
B&L donate to Vision Institute (VCN), 100
Vision Institute internships (VCN), 100
Vision Institute's fund-raising draw winners (VCN), 220
visual fields
Hockey faceguards and visual field scotomas (Wingert and others) (A), 143
A tale of two fields (Williams) (CR), 147

- W -

Wesley-Jessen
W-J Canadian sales skyrocket in 1991 (VCN), 41
Williams TD
A tale of two fields (CR), 147
Unusual presentations of retinal detachment (CR), 73
Wingert TA, Long WF, Martin WG, Hockey faceguards and visual field scotomas (A), 143
Wood G, Iridology show sparks response (L), 118
World's Fair, Seville, Spain
We are awaiting the royalty cheque (VCN), 160

Prévisions financières

Prévisions financières est une rubrique du CJO*RCO *des bureaux d'Ottawa de ScotiaMcLeod Inc, rédigée par un des conseillers en placements de cette entreprise, M. Robert Décarie. Tout avis, commentaire ou opinion figurant à cette rubrique n'engage uniquement que l'auteur et ne reflète pas nécessairement l'opinion de la* CJO*RCO. *On conseille aux lecteurs de consulter un conseiller fiscal professionnel pour obtenir plus d'informations. Vous êtes invités à nous faire parvenir vos commentaires. Si vous voulez obtenir des informations plus détaillées sur un type de placement, prière de nous écrire à l'attention de la* CJO*RCO.

Les titres étrangers : une composante importante de votre RÉR

Pour le passé, beaucoup de détenteurs d'un RÉR se sentaient frustrés devant les règles gouvernementales qui limitaient les placements admissibles à un RÉR aux seuls titres canadiens. Heureusement, le budget promulgué en février 1990 prévoit l'augmentation par étapes de la portion du RÉR qui peut être investie dans des titres étrangers, à raison de 2% par année. Ainsi, à partir du plafond original de 10% le maximum augmentera progressivement pour atteindre 20% en 1994. Même s'il ne s'agit pas d'une augmentation considérable, ce changement peut néanmoins avoir une incidence significative sur la composition et le rendement de votre portefeuille RÉR. En effet, les titres étrangers dans un RÉR peuvent, à long terme, donner un sérieux coup de pouce au rendement global du portefeuille, car d'autres pays en Europe et dans l'Extrême-Orient ont connu une croissance économique plus robuste que le Canada, et leurs devises n'ont rien à envier aux devises nord-américaines. Ainsi, depuis maintenant vingt ans le rendement des titres étrangers devance celui des titres nord-américains. De plus, compte tenu des bouleversements dans l'Europe de l'Est et vu la mondialisation de plus en plus marquée de tous les marchés, il y a des opportunités très intéressantes à saisir à l'étranger.

En outre, le placement à l'étranger permet de profiter des fluctuations des taux de change. Par exemple, si vous achetez un placement libellé en DeutscheMarks, et la valeur de ce placement baisse de 10% lors même que le DeutscheMarks apprécie de 15% vis-à-vis du dollar canadien, vous obtiendrez quand même un rendement de 5%. Par contre, si la valeur du placement baissait de 10% et la valeur du mark diminuait simultanément de 15%, la perte totale pour l'investisseur canadien serait de 25%.

Un autre avantage important d'investir dans les titres étrangers est la diversification que cette stratégie permet. En effet, en mettant une partie de vos placements en dehors de l'Amérique du Nord, vous réduisez vos risques de perte, car votre actif ne sera plus dépendant du contexte politique et économique d'un seul pays ou région. Ainsi, quand l'économie canadienne est en panne, vous pouvez bénéficier des opportunités de croissance qui se présentent ailleurs. Quand on pense que l'actif des sociétés inscrite aux bourses canadiennes ne représentent que 3,1% de la capitalisation totale des marchés mondiaux, il devient très clair qu'une certaine diversification géographique est essentiel pour l'investisseur canadien.

Cependant, l'intervention sur les marchés internationaux peut être très compliquée, sinon impossible pour le particulier non averti. En effet, des dizaines de milliers de titres sont échangés chaque jour sur toutes les bourses du monde et l'obtention des informations nécessaires pour être en mesure de prendre des décisions éclairées peut être difficile et très onéreuse. De plus, comme les règlements diffèrent d'un pays à l'autre, la négociation des titres dans d'autres pays peut être frustrante si l'on n'est pas familier avec les usages du lien. Pour cette raison, la meilleure façon de saisir les opportunités sur les marchés étrangers est par le biais des fonds communs de placement globaux et internationaux. Les fonds globaux investissent à la fois sur les marchés internationaux et internes, alors que les fonds internationaux se limitent aux seuls titres étrangers. Pour choisir un fonds global ou international, recherchez ceux dont les placements sont bien diversifiés et qui jouissent d'une équipe de gestion qui a une longue expérience des marchés internationaux. Vous pouvez également investir dans des fonds limités à un pays spécifique, mais vous vous exposez ainsi aux risques liés aux fluctuations du taux de change et vous vous privez de l'avantage de la diversification offerte par les fonds globaux et internationaux. Avant d'investir dans un fonds limité à un seul pays, il faudrait d'abord que vous soyez sûr que les titres de pays présentent d'excellentes perspectives de hausse.

Enfin, il est très important d'obtenir des conseils d'un professionnel si vous avez l'intention de mettre des titres étrangers dans votre RÉR, car Revenu Canada impose de lourdes pénalités si vous dépassez le plafond établi. Cette pénalité est

l'équivalent de 1% par mois de la valeur de la portion de contenu étranger qui dépasse le plafond. La façon dont le contenu étranger est calculé est également très stricte, étant basée sur le coût d'acquisition des titres en question. Encore une fois, il est important d'obtenir des conseils professionnels.

Emprunter pour investir : ce qu'il faut savoir

L'opinion a toujours été partagée sur l'avantage d'emprunter pour investir, particulièrement en période de détente des taux d'intérêt. A quel moment vaut-il la peine d'emprunter? Même si les règles à ce sujet sont complexes, et la législation passablement stricte, à certains moments il est vraiment avantageux d'emprunter pour faire des placements.

Or, le degré de déductibilité des intérêts que vous payez sur un emprunt dépend de l'usage que vous faites de celui-ci. La règle d'or dit que si vous pouvez prouver que les fonds ont été utilisés pour investir ou pour payer des frais d'entreprise, les fonds seront déductibles. Si vous investissez les fonds, les intérêts seront déductibles seulement si les placements procurent un revenu. Ainsi, si vous avez emprunté pour acheter des marchandises ou des contrats à terme, les intérêts sur votre emprunt ne seront pas déductibles, tandis que dans le cas des actions ordinaires versant des dividendes, ils le seront. De même, les intérêts sur les soldes débiteurs d'une carte de crédit et sur votre prêt hypothécaire ne sont pas déductibles, alors que si vous empruntez pour acheter des obligations d'épargne du Canada par prélèvement salarial, ou si vous utilisez un compte sur marge chez votre courtier, les intérêts seront déductibles. Ceci dit, il y a certaines exceptions. Par exemple, il serait logique que les intérêts sur un prêt pour faire un versement dans un RÉR soient déductibles étant donné qu'il s'agit d'un emprunt pour faire des placements; malheureusement, ce n'est pas le cas: le gouvernement a supprimé cette déduction en 1981.

Le recours à l'emprunt offre plusieurs stratégies de placement intéressants. Par exemple, si votre maison est entièrement payée, vous pourriez la ré-hypothéquer, et investir le produit: dans ce cas les intérêts seraient déductibles. Qui plus est, vous pourriez le faire en puisant les fonds dans votre RÉR autogéré, ce qui vous procure un avantage supplémentaire, car vous ferez ainsi des versements hypothécaires dans votre propre RÉR au lieu de payer des intérêts à une institution financière. Cette stratégie n'est pas pour tout le monde, mais elle constitue néanmoins une des façons d'utiliser le capital que vous vous êtes constitué et qui serait autrement immobilisé dans votre maison.

Une autre stratégie consiste à transformer les intérêts non déductibles en intérêts déductibles. Comment? Si vous avez une hypothèque ou une autre dette non déductible et un portefeuille de placements entièrement payés, vous pourriez vendre ceux-ci et utiliser le produit pour rembourser votre hypothèque ou autre dette. Ensuite, vous n'avez qu'à emprunter des fonds pour racheter des placements dans votre portefeuille, et comme les fonds auront été utilisés pour acheter des placements, les intérêts seront déductibles.

Vous aurez également avantage à minimiser vos dettes non déductibles. Or, cette stratégie est loin d'être un grand coup financier, mais elle peut s'avérer très rentable à la longue.

Considérez par exemple, que pour récupérer des intérêts non déductibles de 12%, il faut réaliser un taux de rendement de 24% sur votre placement!

Enfin, si vous voudrez profiter de la déductibilité des intérêts mais vous n'avez pas de dette non déductible et vous ne voulez pas mettre en cause votre maison, considérez donc la possibilité d'utiliser les facilités de crédit offertes par votre courtier. Les courtiers offrent aux investisseurs admissibles un compte sur marge comportant des taux d'intérêt très intéressants par rapport aux taux courants des autres institutions financières. Cette marge de crédit vous permet de profiter de l'effet de levier sur votre capital, c'est-à-dire qu'avec un montant minimal de capital, vous pouvez réaliser des gains importants. Cette stratégie consiste à emprunter des fonds d'une maison de courtage, qui accepte les valeurs achetées comme gage pour votre dette. Vous devez verser une mise de fonds en espèces (dont le pourcentage varie selon le type de valeur que vous envisager d'acheter) et le courtier vous prêtera le reste. Ainsi, vous avez la possibilité de faire des gains non seulement sur votre propre argent, mais sur les fonds empruntés aussi. Cependant, l'effet de levier peut vous être défavorable dans certaines circonstances, en amplifiant les pertes qui peuvent parfois excéder la mise de fonds initiale. Encore une fois, cette stratégie n'est pas pour tout le monde, et elle est plus risquée que les opérations sur comptant. Cependant, pour certains investisseurs elle représente une façon de réaliser des gains substantiels.

Une des pièges dont il faut se méfier est que les intérêts sur un placement deviennent non déductibles si vous vendez celui-ci pour récupérer les fonds empruntés. Par exemple, supposons que vous emprunter des fonds pour acheter des valeurs de rendement et que subséquemment vous vendez celles-ci pour dépenser le produit à des fins personnelles. Dans ce cas, les intérêts deviendraient non déductibles parce que vous ne possédez plus le

placement à revenu. Pour cette raison, il peut parfois être plus utile de garder un placement pour éviter de perdre la déduction qu'il procure.

Il faut également savoir que le fait d'emprunter pour investir aura une incidence sur votre perte nette cumulative sur placement (PNCP). Votre PNCP à la fin de l'année fiscale représente la somme totale des frais de placement déclarés cette année-là, et toutes les années précédentes depuis 1987 qui excèdent le total de vos revenus de placement au cours de la même période. Quand vous déclarez des frais de placement qui créent ou qui augmentent votre PNCP, vous réduisez d'autant le montant de votre exonération de gains en capital pour l'année en question.

D'autres règles et règlements s'appliquent dans ce domaine. Il vaut donc mieux consulter votre conseiller en fiscalité avant d'entreprendre une stratégie qui implique l'emprunt pour faire des placements, afin d'être au fait de toutes les règles qui pourront avoir des répercussions sur votre stratégie, et pour sûr d'avoir choisi la stratégie qui convient le mieux à votre situation financière.

MON ÉQUILIBRE

MON MILIEU DE VIE

L'ASSOCIATION CANADIENNE POUR LA SANTÉ MENTALE

Financial Foresight

Financial Foresight is provided to the CJO*RCO *from the Ottawa offices of ScotiaMcLeod Inc and written by one of the firm's investment executives, Mr Robert Décarie. Advice, comments and opinions offered in this column are strictly those of the author and are not necessarily endorsed by the* CJO*RCO. *Readers are advised to seek the advice of a qualified tax professional for further information. We invite your comments. If there is an investment concern about which you might wish more detailed information, please drop us a line at the* CJO*RCO.

Foreign assets: An important part of your RSP

Many RSP investors have been frustrated in the past by regulations that generally restrict the investments that can be held in RSPs to Canadian investments. Legislation introduced with the February 1990 budget proposed that the portion of RSP assets that can be invested in foreign holdings will be increased gradually, in 2% increments, from the original level of 10% until it reaches 20% in 1994. Although this is not a huge change, it could make a significant difference to the composition and the performance of your RSP portfolio. Holding foreign assets in an RSP portfolio can result in enhanced performance over the long term because other countries both in Europe and in the Far East have enjoyed stronger economic growth than we have in Canada, and their currencies have also been very strong against North American currencies. In turn, their securities have outperformed those that trade on North American markets over the past 20 years. Also, with the political changes occurring in Eastern Europe and the globalization of the world's economies in general, there are many exciting opportunities for growth to be found other than in North America.

Investing in international markets also affords opportunities for enhancing performance due to currency appreciation. For example, if you were to purchase an investment denominated in German Deutsche Marks and the investment declined by 10% but the German Mark gained strength against the Canadian dollar by 15%, your return would be 5%. If, on the other hand, the investment declined by 10% and the value of the Mark also declined by 15%, the total loss to the Canadian investor would be 25%.

Another major benefit of investing in foreign securities is diversification. By diversifying your investments outside of North America, you are lowering the overall risk of your portfolio because it is not dependent on the economic, political and general circumstances of just one country. Therefore, when Canada's economy is in a slump, there will usually be better opportunities to be found elsewhere. When you consider that the value of companies listed on Canadian stock markets represents just 3.1% of the total capitalization of the larger world equity markets, international diversification makes even more sense for Canadian investors.

Trading in international stock markets by yourself can be complex, if not impossible. Tens of thousands of stocks are traded worldwide, and obtaining the necessary information to make intelligent investment decisions can be difficult and expensive. Also, because regulatory requirements differ from country to

Introducing Visatear.*
Soothing tears of relief for even the driest eye.

A clear solution for eyes with insufficient lubrication.

A sterile, buffered solution, Visatear* is formulated to emulate natural tears, restoring the natural moisture balance of the eye.

With the lubricating action of polyethylene glycol 400, Visatear* acts to protect the surface of the eye, offering highly reliable, effective relief of dry eye.

And Visatear* offers your patients excellent value compared to leading competitive products.[1]

In fact, the patient's first bottle is on us. To receive your complimentary samples, please complete and return the attached sample request card.

To get the dry out

1. Data on file, Pfizer Consumer Health Care, Sept. 1992.

PAAB

* PFIZER CANADA INC. (R.U.) Consumer Health Care Division, Kirkland, Qc., H9J 2M5 (T.M.) Pfizer Inc

Pfizer

country, making trades can be a frustrating exercise. Therefore, global or international mutual funds are an ideal way to invest in these markets, which otherwise are often inaccessible to the individual investor. Global funds invest their assets in securities of foreign countries as well as their own; international funds invest in the securities of other countries but not in those of their own country. When selecting an international or global mutual fund, look for large, well-diversified funds whose management companies have many years of experience in international markets —in both good times and bad. You can also invest in specific-country funds; however, you are exposed to more volatile currency swings and less diversification than you would be with a global or international fund. Before investing in a specific-country fund you should be extremely bullish on the prospects for that particular country.

It is important that you obtain professional advice if you intend to include a foreign component in you RSP, as Revenue Canada assesses penalties if you exceed the allowable limits. This penalty is equal to 1% of the excess foreign content per month. The manner in which the allowable foreign content is calculated is also very strict, as it is based on the acquisition cost of the assets in the RSP. Again, it is important to obtain professional advice.

Borrowing to invest: What you should know

There has always been a great deal of discussion about deductible and nondeductible interest payments, especially in a period of declining interest rates. When does it make sense to borrow and when does it not make sense? Although the rules can be complicated and the government's requirements fairly stringent, there are times when it can make sense to borrow money for investment purposes.

The deductibility of interest payments depends on how you use the borrowed funds. The rule is that if you can demonstrate that the funds have been used for investment for business purposes, the funds are usually deductible. With respect to investments, they must be investments that are expected to produce an income. This means that if you use the borrowed funds to purchase commodities or futures, the interest payments will not be deductible, whereas if you purchase common stock, which pays dividends, your interest payments will be deductible. Other examples include credit card interest payments and mortgage payments that are not deductible, while interest payments to buy Canada Savings Bonds on a payroll deduction plan and interest on a margin account with your broker are. There are exceptions to this general rule, however. For example, one would think that interest payments on funds borrowed to make an RSP contribution would be tax deductible because they are being used for investment purposes, but the government disallowed this practice after 1981.

There are a couple of investment strategies that might be worth considering. One is to re-mortgage a fully paid-for home and use the proceeds for investment purposes, thus making the interest payments tax-deductible. This can also be accomplished using funds from your own self-directed RSP, giving you even more benefits because you are making mortgage payments to your own RSP rather than to a financial institution. While we are not suggesting that everyone who has paid for his or her home run out and re-mortgage it, this is one way to make use of the equity you have built up over the years.

Another strategy to transform nondeductible interest into deductible interest if you have a portfolio of unleveraged investments and a home or other nondeductible debt is to sell off your portfolio and use the proceeds to pay down your mortgage or other debt. Then you could borrow new money to buy back your portfolio and, because you have used the new money to buy investments, the interest payments become tax-deductible.

You should also consider minimizing the amount of nondeductible debt you are carrying. Although this is not an exciting investment strategy, it can be very lucrative. For example, in order to match the return of paying down nondeductible debt that has a 12% interest rate, you would have to earn a 24% rate of return on a fully taxable investment!

Finally, if you do not have any nondeductible debt and do not want to involve your home, but would still like to take advantage of deductible interest, consider using the margin capability offered by your broker. Brokers extend margin to eligible investors at very attractive rates compared with other loan rates available in the marketplace. Using the margin available to you allows you to leverage your investments, which means that you require a small amount of capital relative to the potential gains. This is done by borrowing funds from your brokerage firm and using the securities purchased as collateral. You pay a required percentage of cash for the investment (the percentage varies according to the type of security) and your brokerage firm lends you the rest. By using margin, you have the ability to make profits not only on your money, but on the borrowed funds as well. Leveraging can work the other way also, in that any losses incurred will be

magnified and indeed can even exceed the original cash outlay. Trading is not for everyone and it is definitely more risky than paying cash, but for certain active investors, it can be an attractive way to realise substantial gains.

One pitfall you should be aware of is that interest is no longer deductible if the source of the income it provides disappears. Suppose that you borrow funds to invest and subsequently sell the investment and use the proceeds for personal use. Because you no longer own the income-producing investment, the interest payments are no longer tax-deductible. For this reason, it can sometimes make sense to keep an investment rather than risk losing the tax-deductibility of the interest payments.

You must also consider the fact that borrowing to invest will affect your cumulative net investment loss (CNIL). Your CNIL at the end of any tax year is the total amount of investment expenses claimed in the year and all preceding years since 1987 (when CNIL was introduced) that exceeds the total of your investment income for the same period. When you claim investment expenses that create or increase your CNIL, you reduce your ability to claim a capital gains exemption in that year.

There are other rules and regulations regarding this matter. Consult your tax advisor before you engage in any strategy involving funds borrowed for investment purposes to make sure that you are aware of all regulations that could affect your strategy and to make sure that the strategy you have chosen is appropriate for you.

THE NEW FASHION FAIR FOR EYEWEAR
ALTERNATING WITH OPTICA

optica
optica
optica
fashion

INTERNATIONAL
FAIR FOR FASHION
IN EYEWEAR
COLOGNE
APRIL 23-25, 1993

The distinct fashion fair for eyewear manufacturers and importers as well as their customers, the opticians.
A clear trade fair concept: A selling and ordering fair for eyeglass fashions, concentrating on presentation, information and sales.
A beginning of new objectivity for the fashion side of the industry: The new optica fashion, with all the qualities of the world event, optica.
A concept of new economics through extraordinarily favorable participation conditions and the exclusive offer of specially designed turn-key-booths for exhibitors.
With optica fashion, Cologne is answering to the needs of the industry. And the industry is responding to Cologne.

EXHIBITORS: Please request the exhibiting conditions now for this new marketing event.
OPTICIANS: Please mark the dates for this new ordering fair.
Our office in Canada will assist you in preparing your trip to optica fashion – simply return the coupon below to: Mrs. Edel Wichmann, 480 University Ave., Suite 1410, Toronto, Ontario, M5G 1V2, Tel.:(416) 598-3343, Telefax: (416) 598-1840

KölnMesse

Coupon

Please send me the following information:

❑ Visitor's brochure ❑ Advance registration
❑ Travel package ❑ I would like to exhibit at optica fashion

Kindly attach your business card.

pro fairs
professionals in the trade fair business

Lufthansa

Spectacle's model: Focus-Design-Prize 1992 – Copyright

Vision Care News Actualité oculo-visuelle

(An address for further information, where provided with the source material, is included for each item appearing in Vision Care News.)

Vision Institute's fund-raising draw winners

The Vision Institute of Canada, located on Danforth Avenue in Toronto, has announced the results of its 1992 fund-raising draw and the winners of the $30,000 in prizes. Grand prize winner ($25,000) is Jim Passmore, Toronto. Ancillary prizes were won as follows: $2,500 (Larry Sheldon, Oakville); $1,000 (Samuel Nisenboim, Toronto); $500 (Gary Parsons, Scarborough); $250 (Ian Revill, Cobourg); $100 (E Gillezeau, Pickering; Paul Paoli, Peterborough; Mr and Mrs P Chiarelli, Holland Landing; Paul Dame, Calgary, Alberta; Peter Solo, Toronto); $50 (Vince Panzica, Bramalea; Kirsten North, Ottawa; James Evans, Edmonton; Elio Gualtieri, Orleans; Joan Hansen, Delta, BC).

Our congratulations to all the winners and, from Dr Mitch Samek, a sincere appreciation to everyone who supported the Institute by purchasing tickets in their 1992 fund-raiser.

Canadian wins AOF fellowship

Dr Claude Giasson, currently enrolled in the PhD program at the School of Optometry, University of California at Berkeley, has received one of three prestigious Ezell Fellowships from the American Optometric Foundation (AOF).

The Ezell Fellowship was established by the AOF "to provide incentive and opportunity to talented post-graduate researchers who wish to pursue careers in optometric research and education."

Only some 150 researchers have received Ezell Fellowships since the award was first established in 1949.

Information: Ann Loew, Executive Director, American Optometric Foundation, 4330 East-West Highway, Suite 1117, Bethesda, MD, 20814-4408, USA. Tel (301) 718-6514. Fax (301) 656-0989.

UK optometrist was prominent suffragette

When Victoria Lidiard passed away at the Royal County Hospital, Brighton, England, last October 3rd at the age of 102, she left a double legacy—to the women of Britain and to the profession of Optometry.

In 1912, Victoria (then 23 years old) was imprisoned—in solitary confinement—for her part in a raucous protest on behalf of England's Votes for Women campaign. The protest had included a window-smashing march the length of London's Oxford Street. After hurling a stone through a window in the War Office, she was arrested and found to have a further eight missiles in her pockets. Rather than evade the law, she advised the arresting constable they were "in case the first one wasn't sufficient".

She continued to work on behalf of women's suffrage until the right to vote was extended to the women of the United Kingdom in 1918.

After World War I, she and her husband, Major Alexander Lidiard, trained as ophthalmic optometrists and worked together at the London Refraction Hospital.

Her professional career commitments, however, did not dampen her enthusiasm for the causes of women's rights. She was a member of England's National Council of Women for most of her working life. Even at her death, she was an active proponent and lobbyist for the ordination of women as priests in the Church of England.

Her obituary, published in the October 12 issue of *The Times of London,* concludes that her "defiance and passion lasted with her for over a hundred years; as she saw it, there were some injustices which required more than mere arguments. Fighting for the right to vote was one such injustice; according to Lidiard, it seemed to be 'simply the right thing to do'."

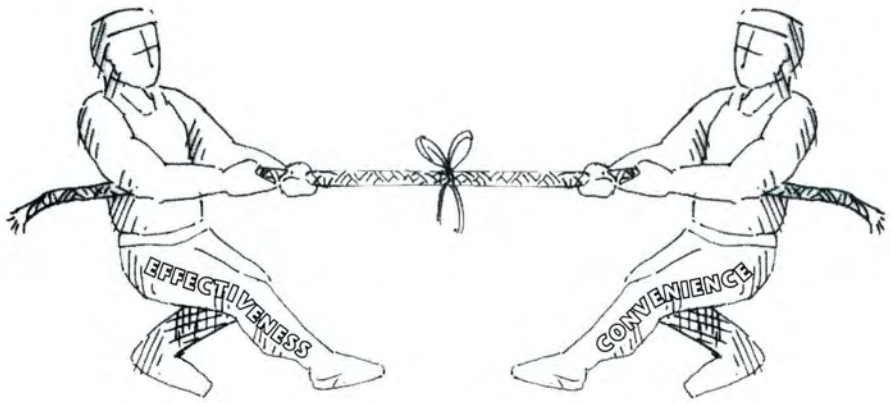
(Editor's note: Our appreciation to CAO President Dr Margaret Hansen des Groseilliers for forwarding Lidiard's obituary to us after reading it while waiting for a connecting flight at London's Heathrow Airport.)

Calgary ophthalmologist named to Alberta Order of Excellence

Dr Howard Gimbel, cited as "an ophthalmologist and pioneer in outpatient cataract and refractive surgery" has been named as a 1992 recipient of Alberta's most prestigious honour, the Alberta Order of Excellence. The announcement was made October 5 by the Hon Gordon Towers, Lieutenant Governor of Alberta. The induction ceremony was held November 26th.

Dr Gimbel is well known to Canadian Ophthalmology and Optometry alike as the founder of Calgary's Gimbel Eye Centre. According to the news release accompanying the announcement, his "advanced cataract and lens implant surgical techniques are accepted as international standards of excellence for their safety and effectiveness".

In addition to the Lieutenant Governor, the governing Council of the Alberta Order of Excellence is made up of six members, including Alberta optometrist and past president of the Alberta Association



MYTH:

"You have to compromise effectiveness for convenience."

of Optometrists Dr Walter Mitson of Lethbridge.

Dr Gimbel has lectured at CAO's Biennial Congress. We are certain that the congratulations of the *CJO*RCO*'s editors are shared by our many colleagues across Canada.

Indian MD wins humanitarian award

Another ophthalmologist, Dr G Venkataswamy of India, has won the Lighthouse Inc's 1992 Pisart Vision Award in recognition of his efforts to prevent and cure needless blindness.

According to a Lighthouse-issued news release announcing the award, as the founding director of India's Aravind Eye Hospitals, Dr Venkataswamy "has saved the eyesight of countless numbers of people by demonstrating the link between vitamin A deficiency and childhood blindness; by devising a hospital system that provides cataract surgery to vast numbers of patients, and by pioneering eye camps that deliver professional vision care to even the most remote villages of India."

The Pisart Award includes a $15,000 prize and is given annually "for the prevention, cure or treatment of vision impairment".

Information: Bonnie Graham, The Lighthouse Inc, 800 Second Avenue, New York, NY, 10017, USA. Tel (212) 808-0077, ext 1424. Fax (212) 808-0110.

Lighthouse calls for 1993 Pisart nominations

The Lighthouse has also issued its call for nominations for the 1993 Pisart Award.

In a recent announcement, the Lighthouse Inc advises, "a candidate may represent any discipline that seeks to prevent or ameliorate vision impairment or blindness including, but not limited to basic scientific research, engineering, medicine, optometry, rehabilitation, or service delivery. The contribution may be a new investigation, invention, or noteworthy finding, or it may represent accomplishments of contemporary relevance that have been made over time."

The Lighthouse Inc is a nonprofit agency dedicated to enabling blind and partially sighted people to lead independent lives. It provides direct services to six facilities in greater New York, conducts practical research, and provides public and professional education and information aimed at benefiting the visually impaired throughout the world.

All nominations for the 13th annual Lighthouse Pisart Vision Award (named after Fernand Pisart, a Belgian diplomat and businessman and funded through the provisions of his wife's, Mme Georgette Pisart, will) must be postmarked on or before April 1, 1993, and sent to The Secretary, Pisart Vision Award Committee, The Lighthouse Inc, 800 Second Avenue, New York, NY, 10017, USA. Tel (212) 808-0077. Fax (212) 808-0110. (This is also the source for further information, including more details on nomination criteria.)

Operation Eyesight

Operation Eyesight Universal is a Scarborough-based charitable organization whose objective is to restore sight to seniors—via cataract surgery—in the developing world. According to a news story in the October 3 issue of the *Toronto Star*, in its 29 years of operation the agency has treated more than 15 million people and helped restore sight to more than 1.2 million eyes. A single donation of $25 will pay all costs associated with restoring sight to one senior.

Donations and information: Operation Eyesight Universal, 759 Warden Avenue, Scarborough, ON, M1L 4B5. Tel (416) 759-8011.

Law firm publishes health bulletin

The fall 1992 issue of *Commentary*, a quarterly information bulletin of legal information, focuses on health law. The eight-page issue includes articles on treating professional partnerships as businesses and investing in real-estate limited partnerships, and, with specific reference to dental partnerships, a summary of the items to cover in a written agreement.

Copies or further information: Fogler, Rubinoff, Barristers and Solicitors, Suite 4400, PO Box 95, Royal Trust Tower, Toronto-Dominion Centre, Toronto, ON, M5K 1G8. Tel (416) 864-9700. Fax (416) 941-8852.

Essilor provides boost to CBMI

Christian Blind Mission International (CBMI) recently announced it will benefit from an expansion of Essilor's continuing support of their mission.

Officially related to the World Health Organization, CBMI is an interdenominational worldwide fellowship dedicated to serving blind, visually impaired and disabled people in the developing world.

The Essilor Group of Companies has decided it will expand its commitment to CBMI through their new Varilux "New Sight with No Lines" program. Under the program, patients who order Varilux No-Line bifocal lenses and donate their old bifocals to CBMI will receive a new, complimentary pair of single vision reading lenses.

Information: CBMI Canada, PO Box 800, Stouffville, ON, L4A 7Z9. Tel (416) 640-6464. Fax (416) 640-4332. Essilor / Canoptec Inc, 375 Deslauriers, St-Laurent (Que), H4N 1W2. Tel (514) 337-0673. Fax (514) 337-2377.

Fight for Sight awards

The Fight for Sight Research Division of the US National Society to Prevent Blindness has announced its 1993–94 Awards Program, including (1) grants-in-aid to fund studies of priority interest and pilot projects (maximum annual award: $12,000); (2) post-doctoral fellowships

REALITY:

"Effectiveness is enhanced by convenience."

With Multi-Purpose Solution, there is no compromise.

Proven Disinfection:

Multi-Purpose Solution contains DYMED, a powerful antimicrobial agent, so selective it doesn't attack ocular tissue. It only attacks and destroys bacteria, fungi and viruses. And unlike peroxide, DYMED doesn't have to be neutralized.

The result? Continuous disinfection that helps prevent recontamination.

Enhanced Cleaning, the Dual Advantage:

Multi-Purpose Solution gives you a dual advantage: unsurpassed power plus the simplicity that helps ensure cleaning frequency.

Unique Convenience and Safety Profile:

Multi-Purpose Solution is simply the most convenient product your patients can buy. What's more, its safety profile is excellent. No wonder Multi-Purpose Solution is Canada's leading lens care product.

Superior Care:

Give your patients the benefit of proven continuous disinfection, a dual cleaning advantage and a unique safety profile.

Multi-Purpose Solution. The new definition for superior care.

BAUSCH & LOMB
Healthcare and Optics Worldwide

Multi-Purpose Solution and Bausch & Lomb are trademarks of Bausch & Lomb Incorporated.

(maximum annual award: $14,000); and (3) student fellowships for summer research ($500 per month for a maximum of three months).

The closing date for receipt of applications is March 1, 1993. Application forms and descriptive brochures are available from Fight for Sight Research Division of the National Society to Prevent Blindness, 500 East Remington Road, Schaumburg, IL, 60173-4557, USA. Tel (708) 843-2020. Fax (708) 843-8458.

News from Bausch & Lomb

Canadians win poster contest

Desmond Fonn, MOptom, and Charline Gauthier, OD, both of the Centre for Contact Lens Research, School of Optometry, University of Waterloo, were the joint winners of Bausch & Lomb's 1992 poster contest held in conjunction with the company's 1992 European Research Symposium in Bordeaux, France. The winning poster, entitled "Dynamic and Static Fluorescein Pattern Evaluation of a Bi-aspheric Back Surface Rigid Lens Design", was selected by a committee established by the International Association of Contact Lens Educators.

Information: Mary Dorton, 1-800-668-7510, ext 207.

Stats from B&L's Olympic screening

Preliminary results from Bausch & Lomb's Olympic screening programs at Barcelona and Albertville have been published.

A key goal for the program was to create a "performance database" against which different levels of athletes can be compared. Combining results from both winter and summer screening programs, the Olympic Vision Centre screened over 700 individuals (nearly 600 of whom were athletes; the balance, coaches and trainers) from 24 different countries at the winter games and from 70 different countries at the summer games.

The Centre also kept stats on patients' histories, their prescriptions and their histories of vision training. Of particular interest to the Centre's practitioners was the fact that only just over half the athletes had ever had a previous eye exam and some 18% (winter) and 33% (summer) reported themselves "currently experiencing visual difficulties". B&L points to this result as clearly indicative of the need for improved vision care for athletes.

Information: Dr Fred Edmunds, Bausch & Lomb / Geneva, Switzerland. Tel 44-22-312-4800. Fax 44-22-312-4840.

B&L launches environmentally friendly packaging

In response to consumer demands to reduce excess packaging, Bausch & Lomb has repackaged two of its most popular brands, Multi-Purpose Solution and Saline, in both cases eliminating the exterior carton completely. The net reduction in the volume of packaging material, accordingly to Larry McGirr, B&L's Marketing Manager of Personal Products, is 38%. In spite of the outer carton's elimination, the new packaging incorporates "an innovative cap and a tamper-evident closure".

New technical consultation line

Bausch & Lomb's Professional Services Department has announced the establishment of a new Technical Consultant's position to answer any of its customers' questions on the company's lenses and solutions and on lens fitting. In a recent news release, the company announced the hiring of consultant Gillian Cashin to inaugurate the service.

The consultation line number is 1-800-263-2642.

B&L's research symposia merge

Next summer (August 20–22), Bausch & Lomb will present a single research symposium in San Diego, aimed at both their customary North American and their European symposia markets.

The company has made a special call for submitted "Free Research" papers from both domestic and overseas locations; authors whose papers are selected will be invited to deliver a 15-minute presentation during the event.

For more information: Gillian Cashin, 1-800-668-7510, extension 305.

CIBA introduces new saline

CIBA Vision has introduced a new saline, which it says compromises neither protection nor comfort at each other's expense.

SoftWear Saline offers a "patented buffer system called ABS (Antimicrobial Buffer System) which is a combination of three borate buffers: boric acid, sodium borate and sodium perborate ... which effectively prevents pathogen growth ... [and] provides excellent, long-term protection for safe lens storage following disinfection."

SoftWear Saline is suitable for all types of soft contact lenses and can be used with heat, chemical and hydrogen-peroxide–based lens care solutions.

Information: John Snisarenko, Product Manager, Lens Care Products,

NO DEPOSIT NO RETURNS

How Multi-Purpose Solution helps keep lenses clean and patients from coming back with problems.

Proven Surfactant Cleaner:

"We conclude that the clinical performance of the Multi-Purpose Solution is equivalent to that of the Opti-Clean II Daily Cleaner", *House, H.O. et al., ICLC Nov-Dec 1991.*

The Frequency Advantage:

Multi-Purpose Solution uses the convenience of its built-in cleaner to enhance effectiveness. Is the increased frequency of use the real secret of our excellent cleaning record? Judge for yourself.

Superior Care:

Give your patients the benefit of proven continuous disinfection, a dual cleaning advantage and a unique safety profile.

Multi-Purpose Solution. The new definition for superior care.

BAUSCH & LOMB
Healthcare and Optics Worldwide

Sensitive Eyes and Bausch & Lomb are trademarks of Bausch & Lomb Incorporated. Opti-Clean II is a trademark of Alcon Laboratories.

CIBA Vision, 2150 Torquay Mews, Mississauga, ON, L5N 2M6. Tel (416) 821-4774. Fax (416) 821-8106.

CIBA invests in Japan

This past November, CIBA Vision became sole shareholder of a major Japanese contact lens company. Ricky Contact Lens, with 1992 sales of about $50 million, is a manufacturer and marketer of contact lenses, lens care products and ophthalmic equipment.

According to Syl Ghirardi, Chief Executive Officer of CIBA Vision Management Inc in Mississauga, the combined sales of Ricky and CIBA will place CIBA in the top three in Japan's Contact Lens market.

Information: Lorraine Johnson, Director, Professional and Clinical Services, 2150 Torquay Mews, Mississauga, ON, L5N 2M6. Tel (416) 821-4774. Fax (416) 821-8106.

Manitoba ODs "unsociable"

Canada's last provincial optometric "Society" has officially changed its name. In mid-November, the former MOS became the Manitoba Association of Optometrists. Question: If they follow in the footsteps of the New Brunswick Association of Optometrists and publish their professional profile under a red cover, does that mean we can anticipate a Canadian edition of MAO's Red Book?

An invitation to all optometrists, exhibitors, spouses and children to attend our CAO Biennial Congress in ***Charlottetown, PEI, August 4–7, 1993.***

It's a chance for optometrists and friends from all across Canada to meet and enjoy such events as an Old-Fashioned Lobster Supper and the musical Anne of Green Gables. *There will be 12 hours of education sessions but also time to enjoy what PEI has to offer—and you may extend your stay and enjoy vacations with your family.*

Register early and save!
Reserve your hotel rooms and flight reservations early!
Contact the CAO office to obtain more information.

CONGRESS HOTLINE: 1-800-663-9815

Tous les optométristes, les exposants, les conjoints et les enfants sont invités à participer au Congrès biennal de l'ACO à ***Charlottetown (Î.-P.-É), du 4 au 7 août 1993.***

Les optométristes et leurs amis de partout au Canada auront la chance de se réunir et d'apprécier des activités comme un dîner au homard à l'ancienne et la comédie musicale Anne of Green Gables. *12 heures seront consacrées à des séances de formation, mais vous aurez aussi le temps de profiter des attraits de l'Î.-P.-É.—et vous pourrez prolonger votre séjour et passer des vacances en famille.*

Inscrivez-vous tôt et épargnez!
Réservez vos chambres d'hôtel et vos billets d'avoion sans tarder!
Pour plus d'information, communiquez avec le bureau de l'ACO.

LIGNE DIRECTE DU CONGRÈS:
1-800-663-9815

The most comprehensive computer system developed ***for optometry.***

OPTOSYS

Version 7.0

does it all!

le fait!

En français / In English

Adapted to each Province
Adapté à chaque province

Parfaitement intégré
Completely integrated

Highly personalizable
Se personnalise facilement

Pour plus d'informations, contactez Patrice au (514) 286-4096.

For more information, please contact Patrice at (514) 286-4096.

O.S.I. Optometric Services Inc. — S.O.I. Services Optométriques Inc.

Le système informatique le plus complet développé ***pour l'optométrie.***

☑ Patient Records/Files
Patient files • Demographics • Records for each optometrist, referral doctors and general practitioners.

☑ Appointment Scheduling
Provides a schedule for each optometrist • User-definable • A week at a glance • Color-coded procedures • Books families & groups easily • Cancellation lists.

☑ Examinations
User-definable • Easy to create personalized exam forms • Data from electronic ophthalmic equipment can be entered directly to the exam form.

☑ Word Processing
Includes most standard editing features • Spell checking • On-line help • Master documents for repeated use.

☑ Tagging/Memos
Keeps a task list for each patient • Daily task lists can be printed to ensure that no service or action is overlooked.

☑ Recalls
Generates recall lists, labels and letters from the patient database • Makes it possible to set the frequency of recalls.

☑ Dispensing
Records all direct patient billings • Processes patient receips & statements • Handles aged accounts receivable • Linked to Inventory module.

☑ Auto-Lens Pricing
Automatically calculates ophthalmic lens and contact lens prices according to specifications • Linked to Patient Records and Inventory modules.

☑ Inventory Control
Creates records for all suppliers • Maintains accurate inventory statistics • Automatic update when purchasing, receiving and returning items • Includes supplier catalogs.

☑ Accounts Payable
Keeps accurate records of supplier accounts • Linked to Inventory module.

☑ MEDSYS™ Medical Billing
Enables electronic telecommunication between your office and provincial health services • Handles all submissions, resubmissions of claims as well as private & third party billings • Verifies billing remittances • Includes choice list of diagnostic codes.

☑ Selection/Report Definition
Enables you to define the reports necessary to ensure accurate and up-to-date record keeping • Highly flexible.

☑ Dossiers Patient
Remplace la filière conventionnelle • Dossiers pour chacun des optométristes, pour les spécialistes et omnipraticiens.

☑ Livre de rendez-vous
Pour chaque optométriste • Flexible • Une semaine entière d'un seul coup d'oeil • Couleur prédéfinie pour chaque type d'intervention • Listes d'attente.

☑ Examen optométrique
Souple et adaptable • Facile de créer des formulaires d'examen personnalisés • Enregistre directement les données provenant d'instruments ophtalmiques.

☑ Traitement de texte
Fonctions d'édition • Dictionnaire orthographique • Fonction d'aide intégrée • Création de documents type.

☑ Notes de service
Conserve une liste de tous les services à faire par rapport à un patient donné • Imprime la liste de tâches quotidienne.

☑ Rappels
Génère des listes de rappel, des étiquettes et des lettres de rappel • Permet de régler la fréquence des rappels.

☑ Facturation aux patients
Rapport complet de toutes les transactions • Traite, classe et met à jour les relevés de compte, les quittances et les soldes • Relié au module Inventaire.

☑ Tarification automatique
Calcule automatiquent le prix des lentilles ophtalmiques et cornéennes en fonction de la prescription • Relié aux modules Dossiers Patient et Inventaire.

☑ Inventaire
Établit des dossiers pour tous les fournisseurs • Gestion des stocks • Mise à jour automatique des achats, réceptions et retours • Catalogue des produits de chaque fournisseur.

☑ Comptes fournisseurs
Maintient un relevé précis des comptes fournisseurs • Relié au module Inventaire.

☑ MEDSYS™ (Facturation à la Régie)
Permet d'établir une liaison électronique directe entre votre clinique et la Régie de votre province • Traite toutes les réclamations, y compris celles de nature personnelle ou auprès de tierce parties • Vérifie les versements • Liste pré-établie de codes de diagnostique.

☑ Rapports et sélections
Permet de définir les rapports qui serviront à maintenir à jour toutes les données de votre clinique • Très souple.

Calendar

Calendrier

For further information regarding events sponsored by the Canadian Association of Optometrists, unless otherwise indicated, contact the CAO office in Ottawa. For events sponsored by a provincial association or society of optometrists, or for international events, contact the hosting organization identified in the item.

1993

February 13–17: British Columbia Association of Optometrists, Continuing Education seminar, Westin Bayshore Hotel, Vancouver, BC

February 15: Prince Edward Island Association of Optometrists, Council meeting

February 19–20: New Brunswick Association of Optometrists, Council meetings, Fredericton, NB

February 20–23: Optical Society of America in co-operation with the American Academy of Optometry: (1) 8th Topical Meeting, "Noninvasive Assessment of the Visual System" (chair: Dr Arthur Bradley, Indiana School of Optometry); (2) 3rd Topical Meeting, "Ophthalmic and Visual Optics" (co-chairs: Dr Melanie Campbell, Waterloo School of Optometry, Dr Jay Enoch, University of California at Berkeley), Doubletree Hotel, Monterey, California. Information:
Optical Society of America
2010 Massachusetts Avenue
Washington, DC, 20036, USA
Fax (202) 416-6100

March 1–7: Vision Awareness Week in Canada / la Semaine de la vision au Canada. Theme: "Vision Safety / Sécurité de la vision"

March 12: Canadian Optometric Education Trust Fund, 1993 Awards Committee meeting

March 13–14: CAO / ACO, Council meeting, Ottawa, ON

April 1–3: Ontario Association of Optometrists, 1993 Annual Meeting, Delta Chelsea Hotel, Toronto, ON

April 8–14: 9th Asian Pacific Optometric Congress (APOC), Hong Kong (related meeting following the Congress in Beijing, People's Republic of China). Information:
Dr Damien P Smith
APOC 1993 Congress Secretary
7 Cookson Street,
Camberwell 3124
Victoria, Australia

April 17–19: Optrafair '93 / Trade Fair of the Ophthalmic Optical Industry of the United Kingdom, National Exhibition Centre, Birmingham, England. Information:
Secretary to the Organizers
Ophthalmic Exhibitors' Association
37/41 Bedford Row
London, WC1R 4JH, England
Tel 071-405-8101
Fax 071-831-2797

April 24: Saskatchewan Association of Optometrists, Council meeting, Saskatoon, SK

April 24–26: Manitoba Association of Optometrists, Annual General Meeting, Westin Hotel, Winnipeg, MB

May 3–8: Alberta Association of Optometrists, TPA Certification Program and OptiFair, Edmonton, AB

May 5: Saskatchewan Association of Optometrists, Council meeting, Saskatoon, SK

May 5–8: Saskatchewan Association of Optometrists, 60th Annual Continuing Education Program, Sheraton Cavalier Hotel, Saskatoon, SK

May 6: Saskatchewan Association of Optometrists, Special General Meeting, Sheraton Cavalier Hotel, Saskatoon, SK

May 15–16: International Contact Lens Conference, Bally's Park Place Hotel, Atlantic City, NJ, USA. Information:
International Contact Lens Conference
PO Box 5876
Harrisburg, PA
17110, USA
Tel (717) 233-8783

May 17: Prince Edward Island Association of Optometrists, Council meeting

May 27–30: British Contact Lens Association, 17th Annual Clinical Conference and Exhibition, Royal Lancaster Hotel, London, England. Information:
Vivien Freeman
Secretary General
British Contact Lens Association
PO Box 1719
London, NW3 4PW, England
Tel (+44) 071-431-0831
Fax (+44) 071-794-0397

May 30 – June 4: British Columbia Association of Optometrists, 1993 Annual General Meeting, Whistler, BC

LUNETTES

GIVENCHY

June 6: Association des optométristes du Québec, Assemblée générale annuelle

June 11–12: New Brunswick Association of Optometrists, Council meetings, Fredericton, NB

August 2: CAO / ACO, Council and Canadian Council of Provincial Presidents meetings, Charlottetown, PEI

August 3: CAO / ACO, House of Delegates (inaugural meeting), Charlottetown, PEI

August 4–7: Canadian Association of Optometrists / Association canadienne des optométristes, PEI Paradise / Paradis Î-P-E, CAO's 23rd Biennial Congress / 23^{e} congrès biennal de l'ACO, Charlottetown

August 21–22: Saskatchewan Association of Optometrists, Golf tournament, Battleford, SK

August 30: Prince Edward Island Association of Optometrists, Council meeting

September 10–12: Ontario Association of Optometrists, 2nd semi–annual meeting

September 11: Saskatchewan Association of Optometrists, Council meeting, Saskatoon, SK

September 15–18: New Brunswick Association of Optometrists, Council meetings and Annual General Meeting, Fredericton, NB

September 24–25: Newfoundland Association of Optometrists, 1993 Annual General Meeting, Albatross Motel, Gander, NF

TBA: British Columbia Association of Optometrists, Okanagan / Kootenays Education seminar

October 2–3: Association des optométristes du Québec, Colloque international sur la lentille de contact, Palais de congrès, Montréal (Qué)

TBA: Manitoba Association of Optometrists, Semi-annual meeting

TBA: CAO / ACO, autumn Council meeting

November 4: Saskatchewan Association of Optometrists, Council meeting, Regina, SK

November 5–6: Saskatchewan Association of Optometrists, 84th Annual General Meeting, Ramada Hotel, Regina, SK

November 7: Saskatchewan Association of Optometrists, Council meeting, Regina, SK

November 8: Prince Edward Island Association of Optometrists, Council meeting

1994

April 30 – May 3: Optica Cologne International Trade Fair for Ophthalmic Optics, Cologne, Germany. Information:
Marianne Hollmann
Messe- und Ausstellungs-GmbH Koln
Messeplatz 1, Postfach 210760
W-5000 Koln 21
Germany

June 23–25: International Medical Contact Lens Symposium, Westin Harbour Castle Hotel, Toronto, ON. Information:
Dr Harold A Stein
President, 1994 Congress
c/o Bochner Eye Institute
40 Prince Arthur Avenue
Toronto, ON, M5R 1A9
Tel (416) 966-3336
Fax (416) 966-8917

1995

summer: Canadian Association of Optometrists / Association canadienne des optométristes, CAO's 24th Biennial Congress / 24e congrès biennal de l'ACO, Calgary, Alberta.

FOR SALE

Optometry practices for sale in
New South Wales, Australia,
one located in the picturesque New England region
and the other in the famous wine-producing
and coal-mining region of the Hunter Valley.
For further information of contact numbers,
please write to
Box 4-2
c/o *CJO*RCO*
Suite 301, 1785 Alta Vista Drive
Ottawa, Ontario, K1G 3Y6

OPTOMETRIC ASSOCIATE

Large, expanding optometric practice requires an optometric associate to practice in beautiful rural Newfoundland. The practice is located near major Atlantic salmon rivers, downhill ski slopes and cross-country ski trails.
Earnings will commensurate with experience but will range between $70,000 and $100,000+.
Candidates must be graduates of an approved School of Optometry and eligible to write the Newfoundland Optometric Board exams or the Atlantic Board exams.

Reply to:
Box 4-1
c/o *CJO*RCO*
Suite 301, 1785 Alta Vista Drive
Ottawa, Ontario, K1G 3Y6

The Practice Builders.

Build your practice on a strong foundation.

These planned replacement alternatives give you and your patients a choice. Choose SeeQuence when you want excellent comfort and visual acuity in a visibility tinted weekly or biweekly disposable.

Choose FreshPak for complete monthly vision care in a single package. This specially designed product has grown to become the most popular planned replacement system in Canada.

Choosing FreshPak and SeeQuence for your practice will help you build it on the strongest foundation of all, patient satisfaction.

For more information, call your Bausch & Lomb representative at 1-800-668-7510.

FreshPak SeeQuence

BAUSCH & LOMB
Healthcare and Optics Worldwide

SeeQuence, FreshPak and Bausch & Lomb are trademarks of Bausch & Lomb Incorporated.

Design Service

We are a design firm that can provide you with an innovative yet functional design solution for your new or present facility. Let us show you how a properly designed facility can project an image of professionalism and success and also make your practice more efficient.

DESIGN • PLANNING • MANAGEMENT

Erindale Designs Limited
60-8 Bristol Rd. East, Suite 234, Mississauga, Ontario, Canada Tel.: (416) 840-0248 Fax: (416) 846-4672 Toll Free: 1-800-665-2021
– FREE ONE TO ONE INITIAL CONSULTATION –

WILLIAM NEWELL SIEBERT
Attorney at Law

307 North Michigan Avenue
Suite 915
Chicago, Illinois
60601, USA
Tel (312) 329-0646

Practice limited to US Immigration law
since 1969

Associate Positions

Available in busy, automated Calgary offices.
Contact Dr Don Bishop for more information.
Income range $85,000 to $110,000 per annum.
Please **write** to:
#620, 11012 Macleod Trail South
Calgary, Alberta, T2J 6A5, Canada
or **phone** (403) 225-0777 (b) or (403) 256-9214 (h)

The Gimbel Eye Centre has immediate openings for Optometric Associates in both Edmonton and Calgary.

Optometrists required

Professional responsibilities will include assistance in pre- and post-operative assessment of cataract patients, counselling and follow-up of refractive surgery patients, and assisting with glaucoma assessments.

The Centre is a private medical facility specializing in out-of-hospital cataract, corneal, laser and refractive surgery.

Please contact Annette Stanwick at **(403)286-3022.**

GIMBEL EYE CENTRE
Suite 450, 4935 - 40 Avenue N.W., Calgary, Alberta T3A 2N1

Full-time Clinical Faculty Position in Optometry

Applications are being accepted for a full-time clinical faculty position in the School of Optometry. Applicants must be eligible for optometric licensure in Ontario. Salary is negotiable within a range commensurate with experience and qualifications. Preference will be given to applicants who have a Residency (specialty) qualification.

General inquiries and/or letters of application with a current resume should be submitted to Dr A.P. Cullen, Director, School of Optometry, University of Waterloo, Waterloo, Ontario, N2L 3G1, Canada.

In accordance with Canadian Immigration requirements, this advertisement is directed to Canadian citizens and permanent residents. The University of Waterloo encourages applications from qualified women and men, members of visible minorities, native people, and persons with disabilities.

Application deadline: February 15, 1993

What Makes ACUVUE® Disposable Contact Lenses The Prescription For Success?

ACUVUE Is Number One In Patient And Practitioner Satisfaction.[1]

Considering the outstanding comfort, visual acuity and convenience of ACUVUE Disposable Contact Lenses, it's really no surprise that ACUVUE is number one in patient satisfaction. And satisfied patients result in fewer problems for you.

But there's more. ACUVUE patients recommend their lenses to four times as many people as ordinary contact lens patients.[2] And ordinary contact lens wearers are more than twice as likely to drop out of their lenses.[3] So satisfied ACUVUE patients mean increased referrals and retention–for a stronger, more profitable practice that keeps growing.

In other words, ACUVUE satisfies your patients. And ACUVUE can help build your practice. Both of which satisfy you. Find out what makes ACUVUE the prescription for success. Convert your practice today.

[1, 2, 3] Independent survey, data on file.

© J&J VPI, 1991

VISTAKON™
Johnson & Johnson VISION PRODUCTS, INC.

MOD. 707 ESPLANO Ph. SERGIO CAMINATA

marcolin®