CJO-RCO

Canadian Journal of Optometry • Revue Canadienne d'Optométrie

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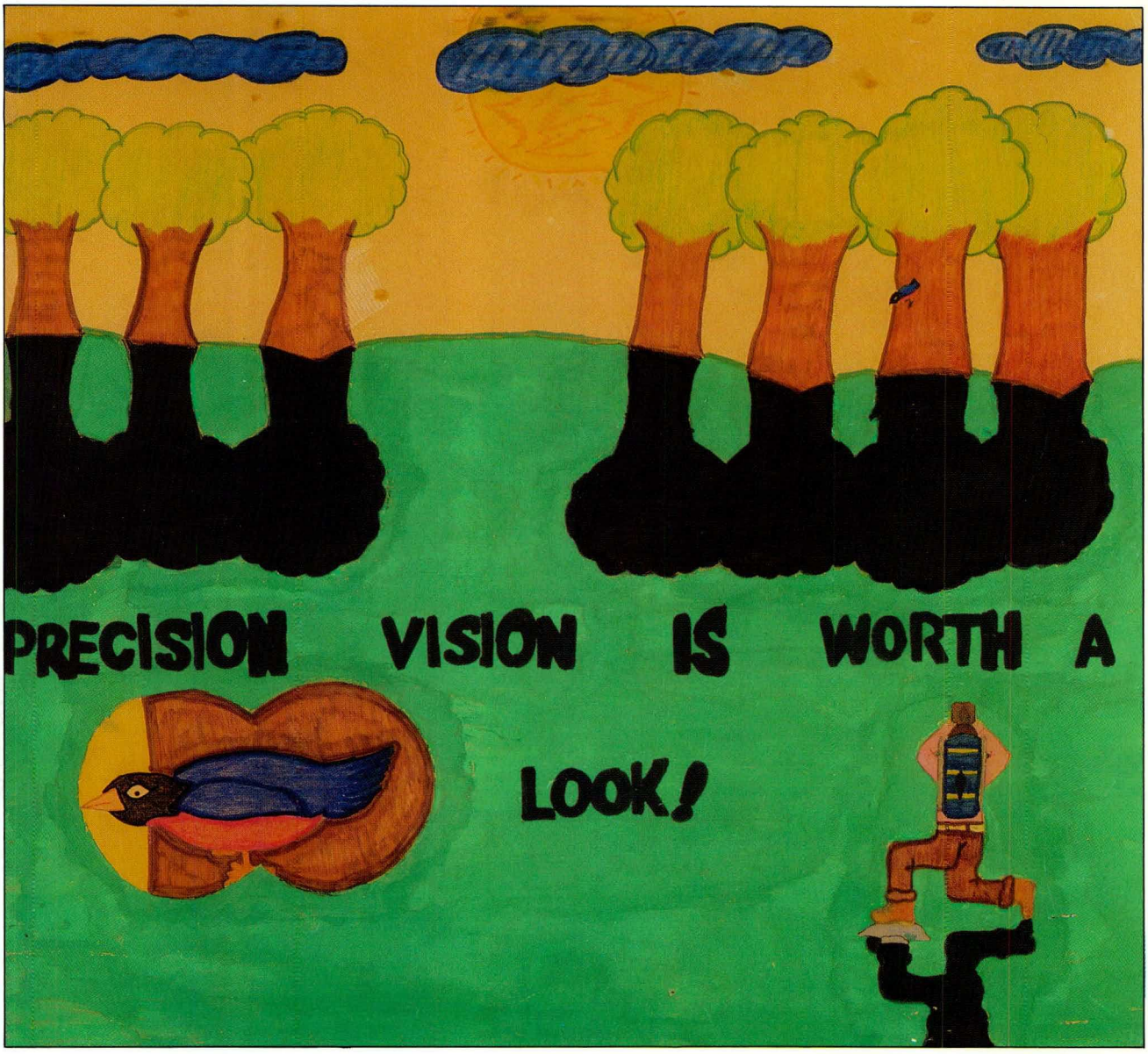

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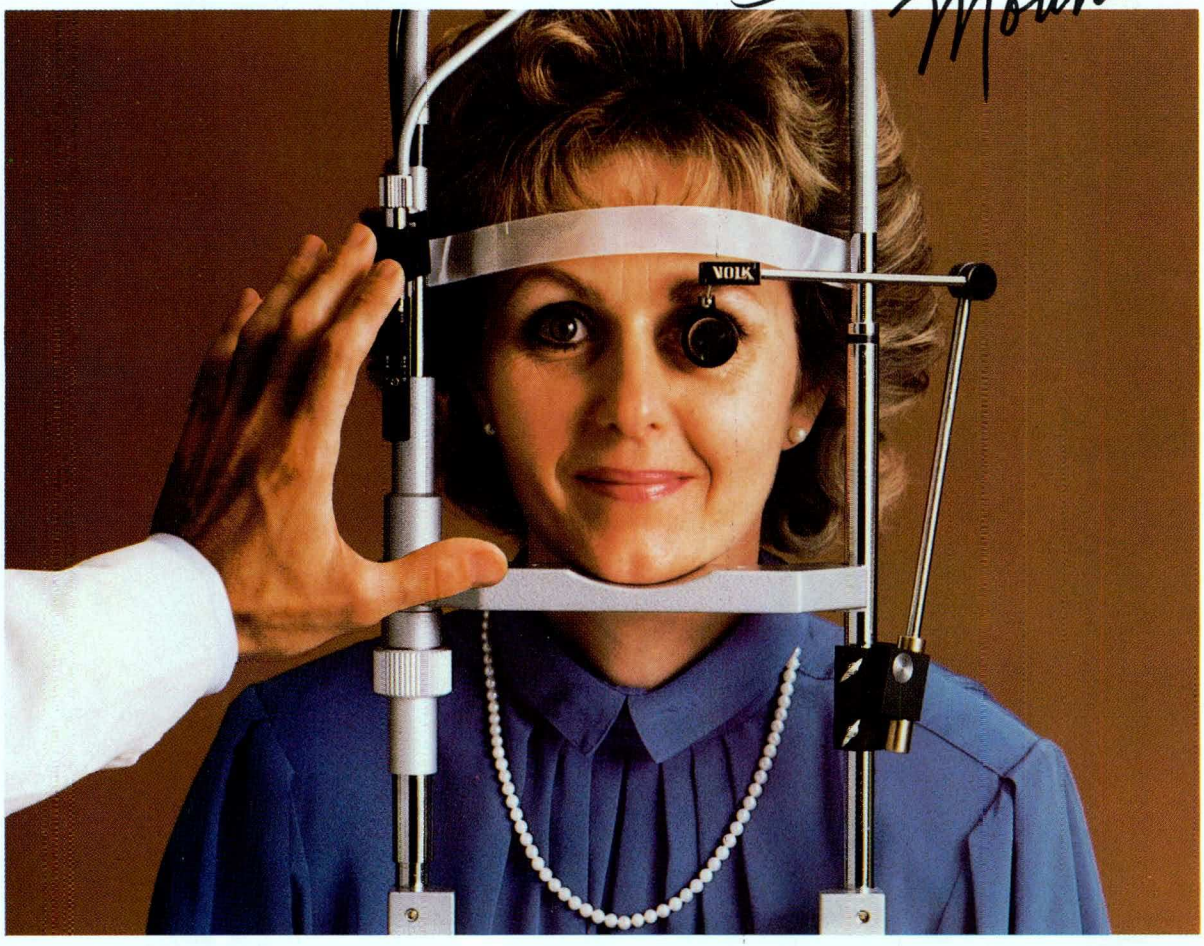

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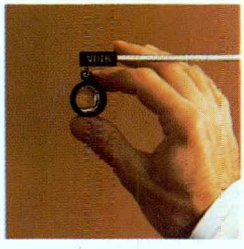

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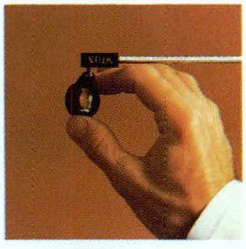

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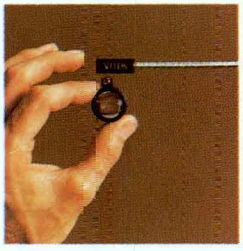

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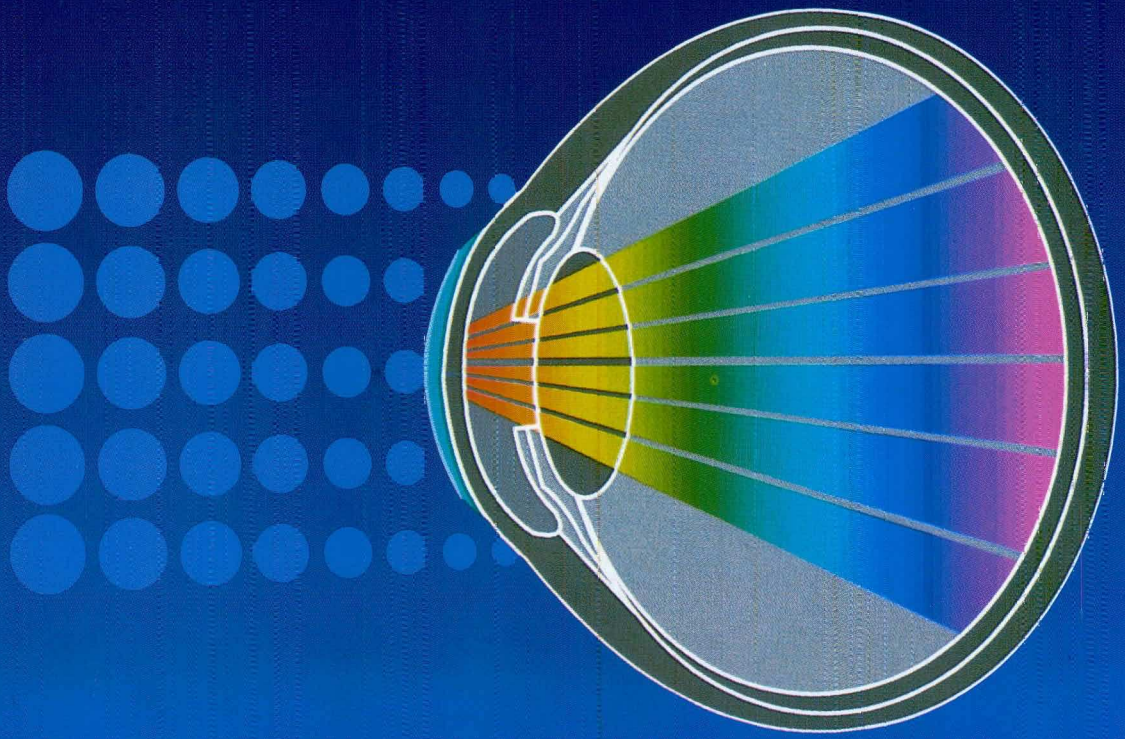

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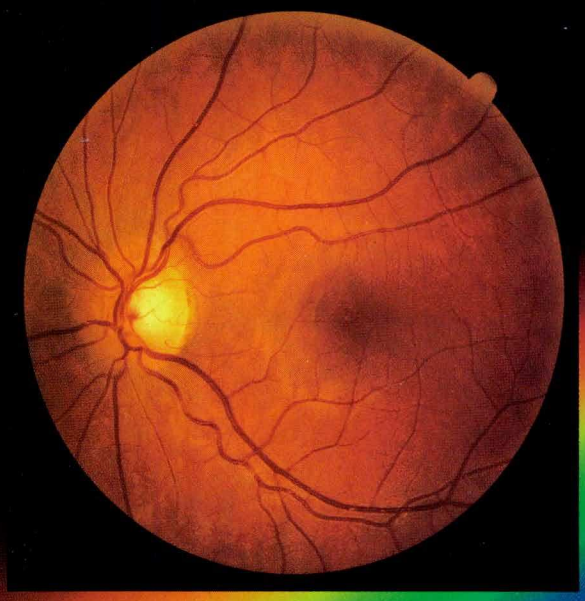

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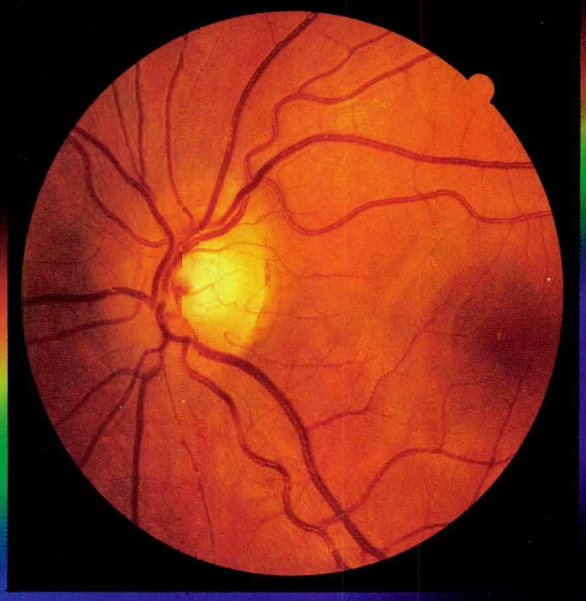

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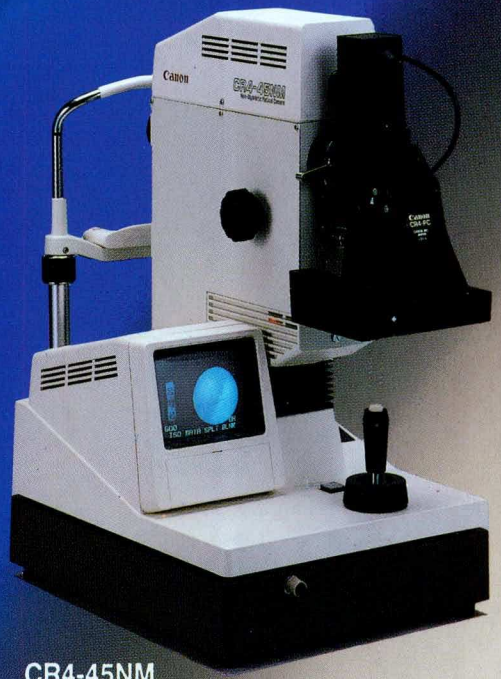

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PRESIDENT'S PODIUM / MOT DU PRÉSIDENT

Such a Vital Message – So Few Messengers

Who amongst us does not believe in the good that Optometry does? Of our 2600 members, the overwhelming majority (100%, I hope!) knows that the optometric profession provides an extremely valuable range of vision care services to 70% of Canadians. But, outside our professional ranks, we instantly become a minority.

If optometrists were the only ones who knew and cared enough to carry the message about the value of Optometry, the messengers would be so small in number, it is unlikely that the majority would hear the message.

What Optometry needs is more messengers — and more effective ones, too.

Recently, I had the opportunity to meet and listen to Tom Sullivan, the star of the autobiographical movie, *If You Could See What I Hear.* Tom has been blind since birth. Yet this fascinating and inspiring man is a Rhodes Scholar, an Olympic wrestler, a 20 handicap golfer, a movie actor who has recently finished his fourth film and just about anything else he wants to be. He has a quick wit and a keen insight that cuts through the haze of preconception and offers a fresh perspective from someone who can't possibly see things as you and I do.

He's a fighter, a winner and he shared some observations about Optometry that make sense to me.

He believes that he and Optometry have something in common, beyond the obvious vision (or lack of vision) connection. He said that, all his life, at whatever he did, he was constantly having to prove himself. He could never expect or experience the luxury of being accepted on past performance or on the assumption that he is capable. He has always had to prove himself and will continue to have to do so throughout his life. "The same," he observed, "can be said of Optometry".

Medical Doctors have monopolized the pedestal of assumed capability for centuries. Optometrists have always had to prove their value and must continue to do so. Perhaps this process is what has made Optometry grow and mature so quickly — over decades instead of centuries. But we must prove ourselves continually. And, along the way, we must educate and inform the millions of Canadians who benefit from optometric vision and eye care of our services' vital importance in the health care field.

The public, perhaps not surprisingly, isn't sure what optometrists do, let alone that we do it so well. But the public does not even assume that optometrists are vision experts! A recent survey done in Alberta showed up some results that astonished me: the Alberta public still doesn't know the difference between the three (or four) "O's": Optometry, Ophthalmology and Ophthalmic Dispensers/Opticians.

(cont't on page 170)

Un message tellement vital – et trop peu de messagers pour le transmettre

Qui parmi nous doute des bienfaits de l'optométrie? Parmi nos 2 600 membres, la très grande majorité (100 %, je l'espère!) connaît la très vaste gamme de services oculo-visuels dispensés par les optométristes à 70 % des Canadiens. Mais, hors des rangs de notre profession, nous devenons subitement une minorité.

Si les optométristes étaient les seuls à connaître le message concernant la valeur de l'optométrie et à veiller à le transmettre, le nombre de messagers serait trop faible, et la majorité aurait peine à percevoir le message.

L'optométrie a besoin de plus de messagers — et de messagers plus efficaces aussi.

J'ai dernièrement eu l'occasion de rencontrer et d'écouter Tom Sullivan, vedette du film autobiographique *Si tu voyais ce que j'entends*. Tom est aveugle de naissance. Pourtant cet homme fascinant et vivifiant est un boursier de la fondation Rhodes, un lutteur olympique, un golfeur qui a un handicap de 20, un acteur de cinéma qui vient d'achever son quatrième film et une personne qui fait tout ce qu'elle désire. Son esprit vif et pénétrant dissipe le brouillard des préjugés et découvre des perspectives nouvelles que vous et moi serions probablement incapables de voir.

C'est un batailleur, un gagnant qui a fait part de ses observations sur l'optométrie, que je trouve sensées.

Il croit que lui et l'optométrie ont un point en commun qui dépasse la vision elle-même (ou le manque de vision). Il affirme que, toute sa vie, en toutes choses, il a dû faire ses preuves. Il n'a jamais eu le luxe de compter sur ses succès antérieurs ni de pouvoir compter sur ses capacités futures. Il a toujours dû faire ses preuves et devra poursuivre dans cette voie toute sa vie. "On peut faire le même observation à propos de l'optométrie", fait-il remarquer.

Les médicins ont le monopole du présumé savoir depuis des siècles. Les optométristes ont toujours été contraints de prouver leur valeur et doivent continuer encore aujourd'hui. Serait-ce pour cette raison que l'optométrie n'a pris que des décennies et non des siècles pour grandir et mûrir? Sans cesse, nous devons prouver nos capacités. Et, dans la foulée, nous devons informer les millions de Canadiens qui bénéficient de soins oculo-visuels de l'importance primordiale de nos services dans le domaine des soins de santé.

La population, doit-on s'en étonner, connaît mal la fonction des optométristes, et encore moins notre grande capacité à nous en acquitter. Mais la population ne soupçonne même pas que les optométristes sont des experts de la vision! Une enquête menée récemment en Alberta montre certains résultats que je trouve étonnants: on n'y fait pas encore la différence entre les

(suite à la page 231)

PRESIDENT'S PODIUM

(con't from page 169)

The majority even ranked opticians and ophthalmic dispensers ahead of optometrists and ophthalmologists as the most knowledgeable sources of information to consult if they have a vision problem! (Tom Sullivan says, however, that I should not be astonished by this.)

We need to spread the message and 2600 optometrists in Canada can't do it alone. So who is going to help?

The minority of the population who knows Optometry's valuable role in the health care system — that's who. They are the optometrists' families; the team of optometric ancillary personnel who daily help provide those valuable services; the representatives of the ophthalmic business related community who have taken the time to get to know their optometric clients and the work they do; those "special" patients; teachers; coaches; other health care workers who have had an opportunity to observe or experience first hand the benefits of optometric care...

These are the people who can help spread our message.

These are the people who can swell the ranks of the messengers tenfold. They are the untapped resource which must now be tapped and mobilized if Optometry's message is to reach a significant part of that vast majority out there.

I am happy to say that one such seed has already germinated in Alberta. The Alberta Association of Optometrists' 1987 Annual General Meeting, held in Calgary in November, saw the creation of the Alberta Chapter of the CAO Optometric Advocates Section. It is my hope that it will grow and flourish and be followed by other chapters springing to life in provinces from BC to Newfoundland.

Alberta was honoured to have the President of the American Optometric Association's Auxiliary, Alana LaRock, come to the inaugural meeting to provide encouragement and helpful tips. Specific examples of successful projects to get Optometry's message across, excellent new audio-visual materials which are becoming available regularly, organizational guidelines, etc., were all discussed.

An Auxiliary, such as the AOA's, can be of great benefit to Optometry. But it suffers from a stifling malady. It is restricted to spouses and thereby eliminates many of the most valuable messengers we have.

It is an image problem, even among those who are spouses of optometrists. As more and more couples are both working professionals and as more and more women enter the profession of Optometry, the thought of joining an Auxiliary, with its "sewing circle" images, however inaccurate they may be, is not very appealing to a great many optometric spouses, male or female.

For these reasons, the Auxiliary concept has been and gone in most areas of this country and the Alberta group was repeatedly reminded that theirs is not an Auxiliary.

We have the opportunity to start fresh, to avoid the problems stifling any Auxiliary, but to build on the great base that the AOA and other Auxiliaries have provided for us in order to create the most effective messenger service ever devised.

(cont't on page 231)

CJO • RCO

EDITORIAL

Improving Our Clinical Skills

It has never been a stated policy of the *CJO * RCO* to offer Editorial comment on the content of a given issue. Only on rare occasions in the past have we done so. However, because of the clinical implications of the technical papers in the Autumn, 1987 issue, we feel that some comment is merited.

Applied optics has always been a strong point in Optometry's training program although, for some years now, an emphasis on pathology recognition and the physiological aspects of contact lens fitting seems to have obscured the therapeutic and clinical value of spectacles.

The paper by Bolduc and Gresset, for example, describing the adapting of the principle of the Franklin bifocal to solve the problems of a paralytic tropia is a case in point. The Franklin bifocal is not a new device, but neither does it become obsolete or useless because of its age.

This type of clinical expertise to solve a problem of convergence has been reported as long ago as our own July, 1971 issue. Likely it could be used more frequently in those cases all too often described by "nothing further can be done".

Children's Vision is a field begging for greater involvement by Optometry, particularly with respect to reading problems and underachieving children. Refractive status is not usually a major impediment as optical treatment is simple and straightforward, but oculomotor problems can be. Measurement of convergence and accommodation amplitudes should be routine procedures. Results have little value unless we have criteria to evaluate our findings and to interpret them in relationship to the child's symptoms.

The paper on accommodation by Woodruff established for the first time accommodative standards for children under twelve. It provides practitioners with a better understanding of the function. This could be enhanced if one were to qualify the recording of the test results. Good results by themselves do not imply efficient performance. We would be better clinicians were we to qualify our results with notes such as "blurs and clears every −.50D increase in power; clearing slowly as the test progresses. Child stumbles in reading. Is this a reading problem or an inability to maintain a clear focus?"

A brief, descriptive note can be a lifesaver in a few months' time when the child returns. What interpretation can be placed on the simple recording "O.D.: amplitude 6.50D"?

Physical exertion and body position have always been known as factors that influence physiological functions in a human being. Up until very recently, little was known of these effects on ocular and visual functions. Today's emphasis on "participaction" and physical exercise, whether aerobic or other form, irrespective of age and health status, gives added importance to the paper by Lovasik et al. on vascular and neural changes during body inversion. The clinical implications are important in the counselling of patients from a preventive aspect.

If one hasn't already done so, one should read each of these papers with attention, keeping preventive optometric care firmly in mind.

GMB

1987 CAO President's Award Presentation

Among the many highlights of the 1987 Biennial Congress, "Merry-Tyme Mingle" last August in Saint John, New Brunswick was the presentation of the CAO President's Award to Dr. Irving Baker, Registrar of the College of Optometrists of Ontario.

The presentation came at the conclusion of formalities held in conjunction with the President's Banquet on Friday, August 7. Dr. Bruce Rosner, immediate past President of CAO, introduced Dr. Baker, who was greeted with a lengthy standing ovation. In accepting the award, Dr. Baker delivered a few "philosophically" thoughtful remarks, after which CAO President Dr. Scott Brisbin read the inscription on the Award.

Following are the highlights from Dr. Rosner's, Dr. Baker's and Dr. Brisbin's remarks made at the 1987 President's Award presentation.

Dr. Bruce Rosner Introduces Dr. Baker

"In researching Irving's contributions to the profession, it wasn't long before I realized that I was writing a book. (Don't worry. You're not getting the book tonight!)

"I find as I'm getting older, however, history becomes more interesting and certainly the curriculum vitae which Irving sent me would make an excellent Index for this book.

"It is titled, *The Irving Baker Story.*

"What struck me at the start are just how many major areas of dedication there are in which he has been involved. Any one of these "roads" that he has travelled would have been a significant career path for any individual man. However, with Irving, we have the 401 — a superhighway.

"To give you a little idea of what I'm talking about, he has been actively playing major roles in our profession in a continuous, unbroken string for forty years. Forty years! (I've been involved in organized Optometry for twenty years and at the thought of forty, all I can say is 'whew!')

"The story, however, still goes on. He is truly an amazing man.

"To start with, he graduated in 1943 from the College of Optometry of Ontario. Upon graduation, he was awarded the George M. Bosnell Medal for Clinical Optometry and this is where one of his many special interests — in Applied Optics — started.

"And he's carried these interests throughout his many years in service to this profession. In fact, and I'm not sure if many of you realize this, he practised his profession first throughout the Maritimes, as a District Consultant in the Medical Corps during the Second World War.

"He then entered private practice in 1946 in Toronto and, in 1947, we see the beginnings of "Irving Baker the faculty member".

"He has been a clinician — Clinical Director at the College.

"He was appointed Adjunct Professor at the University of Waterloo in 1967 and, another interesting fact, still holds that position today. The only faculty person to be at the School longer than him is Professor Emeritus Ted Fisher.

"Certainly his career may have been complete with this. But I'm just getting warmed up!

"He was the youngest and longest lasting President of the Ontario Association of Optometrists. (I believe at the age of 28. That's quite an achievement.)

"But still not enough!

"The Association man was off and running. He then spent six years on the Council of CAO, being at the first CAO Congress in Montréal in 1954. He has attended, in fact, 17 of the 20 Congresses that CAO has held over the years.

"At the time, prepaid health care in Canada didn't include Optometry and Irving was a major force in positioning the profession finally to overcome this but, at the same time, he was a major contributor to establishing Optometry at the University of Waterloo.

"(In fact, that's why he missed one of those Congresses and here's one of many dramatic moments I would include if I were to write the book. He was the one that had to stay behind, waiting for that phone call in 1967, which would confirm the decision to accept Optometry at the University of Waterloo. He's the one that got the call and phoned the President at the Congress that year in Montréal to announce that Optometry was in at Waterloo.

"What about Irving Baker the Registrar — the Registrar of registrars?

"That began in 1949. He was with the Board of Examiners of Ontario, then the Board of Directors of the College from 1961 to 1967 and then became Registrar of the College, a position which he still holds.

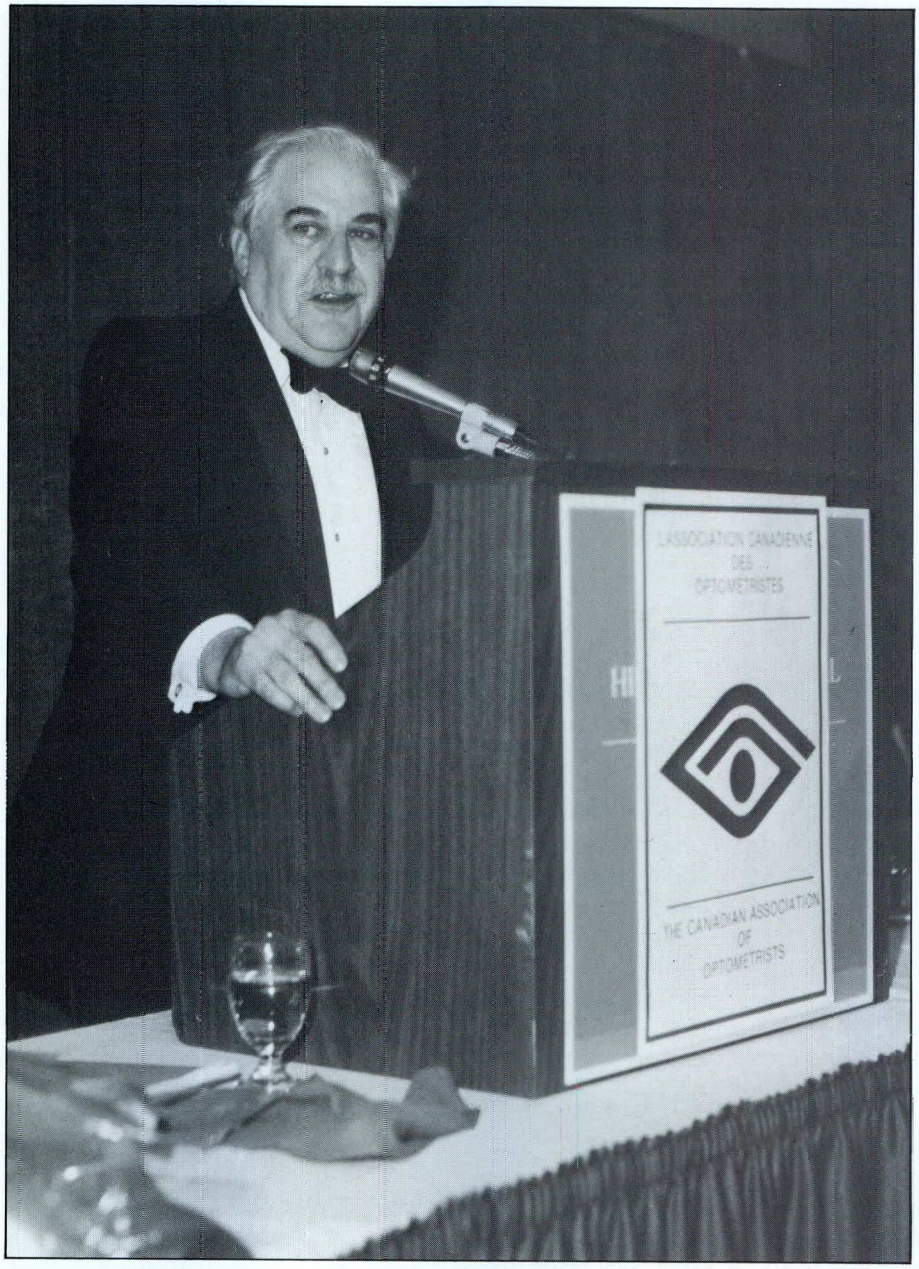
"He became a Fellow of the Academy and in 1949 was on their Editorial Council. He was also Head of their Section on Public Health and Occupational Vision.

"He was made a member Emeritus in 1985, the year after he received the Ontario Association of Optometrists' James Cobean Memorial Award.

"With such a background, it is not surprising to see his name appearing as author or co-author of numerous papers and submissions, for example, to the Commission on Health and the Healing Arts, which eventually led to one of this country's landmark pieces of health legislation, the Ontario Health Disciplines Act of 1974. Today, he continues to author recent submissions to the province's ongoing Health Professions Review.

"He had then, and has now, one of the best optometric minds in the country. And every provincial Association has called him time and time again for his advice.

"I remember many times during my tenure as President and on the Council of the Manitoba Optometric Society when



Dr. Irving Baker

the word would go out — 'Call Irving!' That's all that had to be said. We needed an opinion; we needed some advice; the wording wasn't right; we needed that special insight. 'Call Irving!'

"And the advice was always there. But more than being just an advisor, Irving has an uncanny ability to identify future trends. Put this together with his insistence on excellence, the right word, the right answer and this is what helped produce the many far-reaching advances in this profession across the country and especially in Ontario. No matter how hot the discussions and negotiations got, his remained the coolest head in Canadian Optometry.

"But, through all this, Irving Baker is a strong family man with his wife of nearly forty years, Helen, and their two sons, Mitchell and Jim. In fact, I don't know how Helen has survived those forty years, those many years. (I said I wouldn't say this, Helen.) But she told me the other day when I was talking to her that Irving is an extremely disciplined man, except of course, when it comes to time and, during these long, long meetings, she knew that everything was alright so long as she didn't get a ransom note.

"I don't know if she's ever told you that, Irving.

"The last interesting little tidbit, by the way, is that, back in the early years of the CAO, when funds were rather scarce, the Baker household was the place where many of our CAO Councillors had dinner during their meetings. And, while I wasn't there personally, it's nice to be able to finally return the favour and host the two of you tonight as our guests.

"Well, I'm exhausted, just from having reviewed *The Irving Baker Story*."

Dr. Baker's Acceptance of the 1987 CAO President's Award

"This time of the night is no time to become terribly serious, nor to talk for any great length of time.

"As a matter of fact, you heard some marvelous things about my wife, but I think that it went far beyond the call of duty this afternoon when she sprained her ankle. (It was this afternoon, you see, that I had set aside to write my speech and I never got around to it.) That's rather typical of her contribution, by the way.

"Tonight is full of memories for me because it was some forty-four years ago, just about this time of the year, that I got off a train in Halifax after riding on it for some two days and two nights.

"It was two o'clock in the morning and I arrived at the Halifax station which was rather empty — this was 1943. It was raining, and 'fogging', just what I had to get used to and, as I looked around, carrying all my kit, plus an opththalmoscope and a retinascope, I found one person in the station. He looked at me and I looked at him and I asked where the military police were because I thought maybe they knew where I should be going — because I sure didn't. He asked me who I was, and what I was, and my very first Eastern Canada greeting was, "By God, not another Upper Canadian."

"This used to be the 'Maritime' provinces; they're now the 'Atlantic' provinces and I would wager that in the some three years that I served here that I've probably seen as much or more of all the places you do not want to see in the Atlantic provinces. That was my job — to go everywhere where nobody else wanted to go.

"(Incidentally, the music tonight was very reminiscent of that time and I'm not sure that it was put on for me and my wife, but we appreciated it, nevertheless).

"People who do the kinds of things I've done in four decades are not terribly unusual. In part, we are probably opportunist in the sense that opportunities arise and one takes them. But I must say to you that I've probably gained more than I've given.

"I see a lot of people here who I recognize from when you were students. The only difference is that you still look a lot

younger all the time and many of you I don't recognize at all, likely because, as students, I never found you dressed this well.

"It's been a great time; great, because it's been challenging. It's had its rewards; it's had its frustrations but not very many defeats, because you just don't let frustrations and losses ever amount to a defeat.

"I think I can now talk a little philosophically and identify — or try to identify — where the challenges are going to be for you in the next forty years.

"I was impressed with the CAO meeting today and particularly by the speakers who represented the American Optometric Association and the IOOL. Listening to them, you can come up with a relatively clear picture of the direction in which not only Optometry is going, but in which health care services are likely to go in the next little while.

"I would make a point to you, however. Optometry suffers from a little bit of paranoia, sometimes a whole lot of paranoia. I say that in this context: that we react sometimes as though what is happening to us never happened to anyone else or is happening to anyone else. And that's simply not a fact.

"I've had the opportunity of working with the other four, senior professions, Medicine, Nursing, Dentistry and Pharmacy, for a good number of years now in the province of Ontario and we have a bit of a club, Registrars do. The thing that I learned mostly through those meetings has been that almost all of those groups have problems similar to those of optometrists.

"And I say this because if we continue to suffer feelings that we're persecuted or singled out, I think that our reactions, instead of being pro-active, will be reactive, and that's deadly.

"It's deadly because it stifles dialogue.

"The challenge that faces Optometry and the other health professions I think is pretty clear, at least in Canada.

"We have gone through a phase of evolution from the time we began in the early 1900's in this country, where the only protection that the public had was the licensing process and the law of negligence — which doesn't work. More recently, we've seen an update of licensing and some accountability. But we also see another trend occurring now and accountability without quality assurance just doesn't fly anymore.

"So it seems to me that the challenge lies in several areas and the one thing that I would leave with you is that the most important thing for optometrists and optometric organizations to do is to begin talking to each other. And I don't mean talking in the sense of who's to blame, and who's not to blame, or who's successful or who's not successful. I'm talking about dialogue in a more functional and philosophical sense.

"What are we? Where do we want to go? How do we want to get there? What does the public need?

"Don't worry about who's going to do it. While there seems to be a great deal of emphasis upon communication, which is essential, the fact is that you must have something to communicate. The fact is that, unless you **have** the facts and unless you provide what you say you are providing, it won't fly no matter how good the communications are.

"I think this is a time for you, in granting this honour to me, to stop and think — not about me, but what do **you** want for yourselves, as practitioners; what kind of contribution do you wish to make? I think those are the challenges.

"So on behalf of my family and wife, who really didn't have to fall down and cut this short, thank you."

Dr. Brisbin Concludes the Ceremony

"Irving, I know that not only the people in this room, but optometrists and anyone involved with Optometry all across this country share the sentiments that were expressed here tonight in honouring you, and the debt of gratitude that we all owe to Irving Baker for a great deal of what this profession is today.

"This plaque says,

'The Canadian Association of Optometrists President's Award, presented to Dr. Irving Baker, August 7, 1987 in recognition and sincere appreciation for his tireless commitment as clinician, educator, author, legislator, registrar, advocate and mentor to the profession of Optometry.

'This award pays particular tribute to his dedicated service, motivation and far-reaching influence on the practice of Optometry across Canada.'

"I can't think of any better way to say thank you to Irving Baker."

Corneal Vascularization in a Group of Soft Contact Lens Wearers: Prevalence, Magnitude, Type and Related Factors

J.D. Jantzi *
W.E. Jackson **
K.M. Smith†

Abstract

Groups of 827 soft contact lens wearers and 900 non-contact lens wearers were examined for corneal vascularization by means of biomicroscopy. Slit-lamp photographs were taken to record cases of vascularization and to permit measurements of the magnitude of the vascular response. The prevalence of corneal vascularization in the soft contact lens group was 33.9% compared to 2.0% in the non-contact lens group; a highly significant difference [X^2=304.12, df=1, $p<0.005$]. Of the soft contact lens related vascular responses, 98.6% were located in the superficial stroma, indicating that the conjunctival circulation is most often involved in corneal vascular responses to soft contact lenses. Vascular penetration radially into the cornea from the limbus did not exceed 4.5 mm in either the study or control groups; 96.8% of the vascular responses being 1.5 mm or less in the soft contact lens group. Age and sex of the patients were not significantly related to the observed frequency of corneal vascularization in either the study or control groups. However, refractive error, daily wearing time and the total length of time soft contact lenses had been worn were significantly related to the observed frequency of corneal vascularization in the soft contact lens group.

Even though sampling error and bias affect this type of study, it appears that corneal vascularization is a frequent ocular response in patients who are wearing daily-wear and extended-wear soft contact lenses. This study supports the recommendation that all soft contact lens patients, including those who may be asymptomatic, should be carefully examined at regular intervals for signs of adverse ocular responses such as corneal vascularization.

Introduction

Corneal vascularization is an adverse ocular response to contact lenses. Both PMMA and hydrophilic soft contact lenses have been implicated in the development of superficial and/or deep stromal corneal vascularization in humans as well as in experimental animals.[1-11] In most cases, the zone of vascularization is limited to the peripheral cornea,[12] while in more severe cases, blood vessels and granualation tissue extend to the central optic zone of the cornea.[13]

The physical, physiological, biochemical, neurological and associated factors which are involved in the pathogenesis of corneal vascularization are still incompletely understood. Nevertheless, several factors have been shown to be common to both corneal vascularization and contact lens wear: corneal hypoxia, corneal edema, limbal injection, increased peripheral corneal lactate concentration and immunological responses.[14]

In a study of steep, tight-fitting hard and soft contact lenses, Tomlinson and Haas[15] discovered a direct relationship between the degree of central corneal edema and the degree of limbal vessel injection when corneal edema exceeded 6%. This correlation was noted for hard and soft contact lenses alike, although the former did not impinge physically upon the limbal vessels. It was accordingly suggested that the limbal vascular injection resulted from tissue hypoxia and ensuing corneal edema rather than from mechanical or physical lens factors.

Comparing the limbal vascular responses related to hard and soft contact lenses, McMonnies, Chapman-Davies and Holden[16] reported that soft contact lens wearers had significantly more limbal vascular injection than hard contact lens wearers, even though the average number of years of wear was only 3.8 years for soft lens wearers and 11.6 years for hard lens wearers. They voiced concern that the limbal injection of soft contact lens wearers might ultimately result in corneal vascularization.

McMonnies[17] reviewed anatomical criteria and clinical methods of examining the limbal vasculature for signs of abnormalities prior to contact lens wear in

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order to permit an accurate assessment of the vascular response to the contact lenses later. He stressed the importance of monitoring the limbal vascular response at regular intervals after prescribing contact lenses in order to gain an overall indication of the contact lens tolerance, as well as to allow improved lens fittings to be made before corneal vascularization could develop.

The examination of the zone of capillary end-loops to diagnose corneal vascularization demands careful observation and attention to anatomical detail. Duke-Elder[18] writes:

> "These capillary end-loops do not normally extend beyond the serrated rim of the normal limbal opacity but lie along the line where the limbal curve changes to the corneal. . . . If they transgress the limit of the limbus as a further series of vascular loops, the vascularization is pathological."

McMonnies, Chapman-Davies and Holden[16] stressed the importance of distinguishing between the translucent limbal tissue surrounding the cornea and the transparent corneal tissue by means of retro-illumination techniques when evaluating the limbal capillaries with the biomicroscope. It is known that this zone of translucent limbal tissue is most prominent superiorly and is quite variable in width from person to person, ranging from zero to approximately 2.5 mm from the margin of the opaque sclera. Normal limbal capillary loops are routinely observed within this variable-width translucent limbal zone (Fig. 1), whereas abnormal corneal vessels are observed more axially in clear corneal tissue (Fig. 2). The morphological structure of the limbal loops is also helpful in the early diagnosis of corneal vascularization, for it is known that vascular "spiking" occurs in the initial stages of superficial corneal vascularization[17] (Fig. 3). As the vessels continue to grow into the superficial corneal stroma, they become increasingly tortuous, bifurcate and often become dendritic in appearance (Fig. 4). The diagnosis of deep stromal corneal vascularization is simpler to make since these vessels emerge from deep within the corneal stroma, are straighter than superficial vessels and are discontinuous with the conjunctival circulation (Fig. 5).

The prevalence of corneal vascularization in the population of soft contact lens wearers is unknown and may be impossible to discover exactly because of the difficulties inherent in studying such a large, diverse group. Nevertheless, examining a sample of this population would produce statistical estimates within the limits of sampling error and bias. In addition, data could also be gathered indicating the frequency and type of corneal vascular responses (superficial or deep stromal), the magnitude of vascular infiltration into the cornea, and the relationship of other likely correlated clinical factors such as age, sex, refractive error, daily wearing time and total contact lens wearing time.

Purpose

The purpose of this investigation is:

Part I:
to compare the prevalence, magnitude and type of corneal vascularization in a group of soft contact lens wearers with that of a control group of non-contact lens wearers, and

Part II:
to determine if a correlation exists in soft contact lens wearers between corneal vascularization frequency and the following factors: age, sex, refractive error, daily wearing time and total wearing time.

Method

The study group was comprised of soft contact lens patients who had been

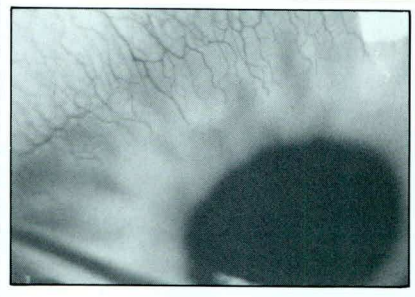

Figure 2
Abnormal corneal vessels outside the zone of normal limbal translucency in clear corneal tissue (96×).

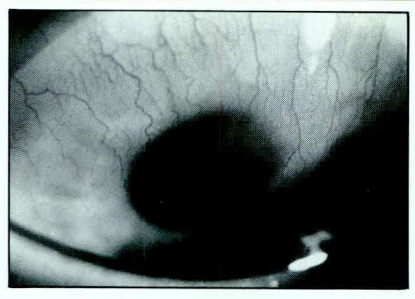

Figure 4
Late stage of superficial stromal corneal vascularization showing increased tortuosity, bifurcation and dendritic appearance (96×).

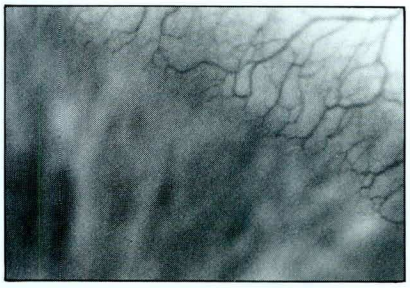

Figure 1
Normal limbal vessels within the variable-width translucent limbal zone surrounding clear corneal tissue (96×)

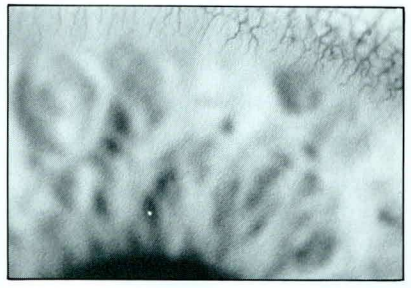

Figure 3
Early stage of superficial stromal corneal vascularization showing vascular "spiking" (96×).

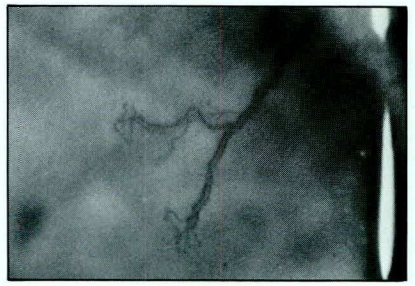

Figure 5
Deep stromal corneal vascularization showing straighter appearance of vessels emerging from deep within the corneal stroma (96×).

wearing daily-wear or extended-wear lenses on a regular basis for at least one year. Soft lens wearers who had previously worn hard contact lenses were exluded from the study. There were 262 males and 565 females in the study group, for a total of 827 soft lens wearers. Lenses were worn on a daily-wear basis by 784 patients while 43 patients were extended-wear.

The control group was made up of patients who had never worn contact lenses. There were 374 males and 526 females in the control group, for a total of 900 non-contact lens wearing patients. Figs. 6–7 and Table 1 show the patient frequency distributions for the study and control groups. The study group ranged in age from 4–75 years with a mean of 28.9 years and a standard deviation of 8.9 years. The control group ranged in age from 8–78 years with a mean of 34.2 years and a standard deviation of 16.3 years (Table 2).

The age frequency distributions of the study and control groups are shown in Figs. 8–9 and Tables 3–4. As might be expected of a group of contact lens wearers, the majority (756/827 or 91.4%) were less than 40 years of age. In the control group, only 580/900 or 64.4% were under the age of 40 years.

The study and control groups consisted of patients who were examined on a daily basis from the patient population of the authors' practice. It was consequently impossible to conceal from the examiners the identity of the group, either study or control, to which the patients belonged.

Prior to the clinical examination, a questionnaire was completed by the soft contact lens wearers which documented their age, sex, refractive error, average daily contact lens wearing time and the total number of years soft contact lenses had been worn. Patients in the study group who had received their contact lenses outside the authors' practice were often unfamiliar with the manufacturer-type, design-type or water content of the soft lenses they were wearing; nor was it always possible for the examiners to determine this information. For these reasons, it was not possible to examine the connection between the frequency of corneal vascularization and manufacturer-type, design-type or water content of the soft contact lenses which the study patients were wearing.

Biomicroscopic examination of the limbal vessels in each of the four quadrants of both eyes was performed using direct and indirect retro-illumination

TABLE 1
Patient Frequency Distributions

Group	Males	Females	Totals
Study	262 (31.7%)	565 (68.3%)	827 (100.0%)
Control	374 (41.6%)	526 (58.4%)	900 (100.0%)

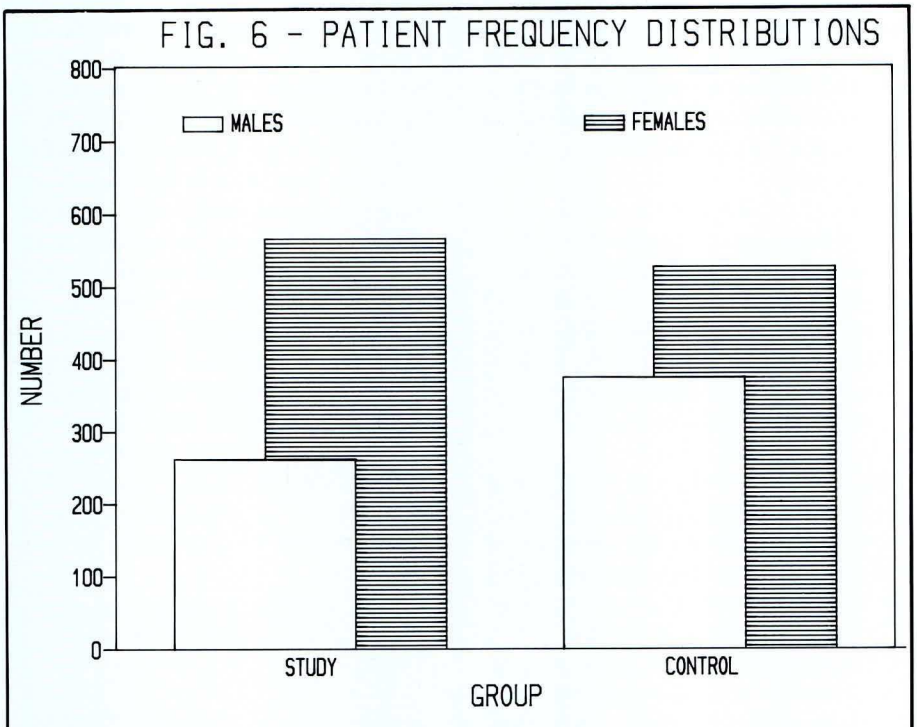

Figure 6
Number of patients in the soft contact lens (study) group and the non-contact lens (control) group by sex.

TABLE 2
Patient Age Statistics (Years)

Group	Range	Mean	Std. Dev.
Study	4–75	28.9	8.9
Control	8–78	34.2	16.3

TABLE 3
Age Frequency Distribution (Study Group N=827)

Age Group	Males	Females	Totals
0–9	0 (0.0%)	1 (0.1%)	1 (0.1%)
10–19	44 (5.3%)	80 (9.7%)	124 (15.0%)
20–29	126 (15.2%)	251 (30.4%)	377 (45.6%)
30–39	70 (8.5%)	184 (22.2%)	254 (30.7%)
40–49	17 (2.1%)	36 (4.4%)	53 (6.4%)
50–59	3 (0.4%)	10 (1.2%)	13 (1.6%)
60–69	1 (0.1%)	2 (0.2%)	3 (0.4%)
70–79	1 (0.1%)	1 (0.1%)	2 (0.2%)
	262 (31.7%)	565 (68.3%)	827 (100.0%)

TABLE 4
Age Frequency Distribution
(Control Group N=900)

Age Group	Males	Females	Totals
0-9	1 (0.1%)	2 (0.2%)	3 (0.3%)
10-19	79 (8.8%)	109 (12.1%)	188 (20.9%)
20-29	63 (7.0%)	95 (10.6%)	158 (17.6%)
30-39	87 (9.7%)	144 (16.0%)	231 (25.7%)
40-49	89 (9.9%)	111 (12.3%)	200 (22.2%)
50-59	43 (4.8%)	45 (5.0%)	88 (9.8%)
60-69	9 (1.0%)	17 (1.9%)	26 (2.9%)
70-79	3 (0.3%)	3 (0.3%)	6 (0.6%)
	374 (41.6%)	526 (58.4%)	900 (100.0%)

TABLE 5
Prevalence of Corneal Vascularization For Each Group
(Study Group N=827) (Control Group N=900)

Group	Males	Females	Totals
Study	97/262 (37.0%)	183/565 (32.4%)	280/827 (33.9%)
Control	7/374 (1.9%)	11/526 (2.1%)	18/900 (2.0%)

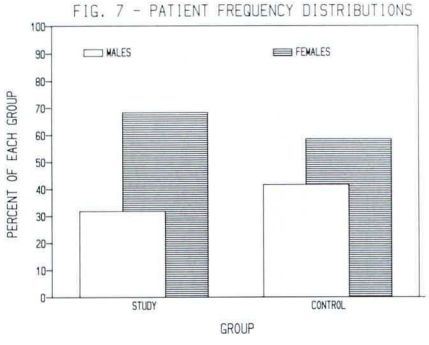

Figure 7
Percentage of patients in the soft contact lens (study) group and the non-contact lens (control) group by sex.

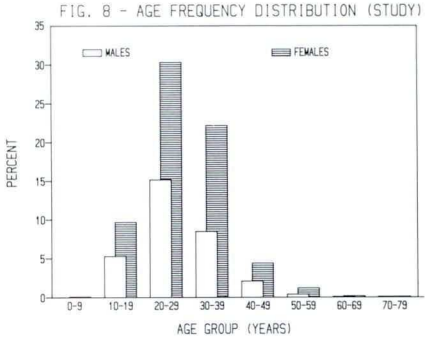

Figure 8
Percentage of patients in the soft contact lens (study) group by age and sex.

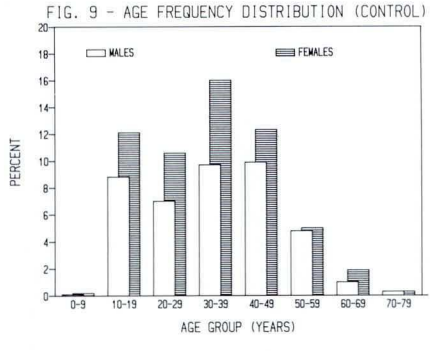

Figure 9
Percentage of patients in the non-contact lens (control) group by age and sex.

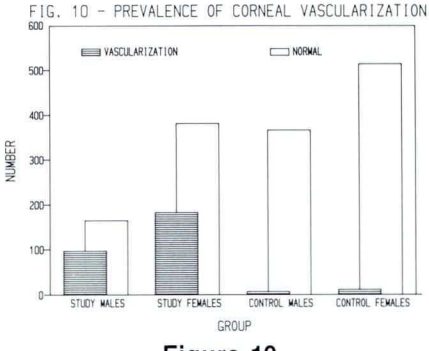

Figure 10
Number of patients by sex in the soft contact lens (study) group and the non-contact lens (control) control group with and without corneal vascularization.

methods. Slit-lamp magnifications ranging from 15–40× were used in conjunction with white and red-free illumination to examine the zone of limbal translucency and the capillary end-loops. Vessels which were observed to extend inward from the edge of the normal zone of limbal translucency into clear corneal tissue were classified as abnormal. In cases where there was no apparent translucent zone, vessels which were observed in clear corneal tissue against the background of the iris were considered abnormal.

Corneas in which vascularization was observed were photographed using a Topcon SL-5D photo slit-lamp and Kodachrome ASA 64 color transparency film. The maximum vessel penetration distance into the cornea from the limbus (the magnitude of the vascular response) was determined later by analyzing projected enlargements of the transparencies; using a comparator to make measurements to the nearest 0.1 mm. The inner margin of the translucent limbal zone, when present, or the periphery of the visible iris was used as the standard reference point for all measurements. Slide analysis magnifications varied from 15–96×.

The type of vascularization, superficial and/or deep stromal, was predetermined at the time of the clinical examination by evaluating the depth of the vessels within the corneal stroma using the usual optic section technique.

Results

Part I: Prevalence, Magnitude and Type of Corneal Vascularization Prevalence

The prevalence of corneal vascularization, as shown in Figs. 10–11 and Table 5, was 33.9% in the soft contact lens group and 2.0% in the control group of non-contact lens wearers. Chi-square analysis showed this difference to be highly significant [$X^2=304.12$, df=1, $p<0.0005$].

Of the 18 cases of corneal vascularization in the control group, 7 resulted from previous corneal infections, 3 from past ocular injuries and 8 were of unknown causes.

Magnitude

The extent of vessel penetration distance into the cornea from the limbus did not exceed 4.5 mm in either the study or control groups (Tables 6–7). However, a

difference was noted in the frequency distributions of the two groups (Figs. 12–13). Whereas 96.8% of soft contact lens wearers had vessel growth within 1.5 mm of the limbus, only 44.4% of the control group were within this region, all of the latter being of unknown causes. Of the known causes of corneal pathology in the control group, (7 keratitis and 3 ocular injuries) all had vessel penetrations 2.0 mm or greater into the cornea. The soft contact lens wearers had vascularization frequencies which appeared to decrease in an exponential manner as a function of vessel penetration distance from the limbus, in contrast to the non-contact lens wearers who had an essentially constant vascularization frequency.

Type

Corneal vascularization was observed in the superficial and deep stromal layers in both groups (Fig. 14; Table 8). Most vascular responses were located in the superficial corneal stroma for soft lens wearers and non-contact lens wearers alike; 98.6% vs 66.7% respectively. Although Chi-square analysis could not be employed because of low expected frequencies (E<5), Fig. 14 does show an obvious preponderance of superficial stromal vascularization in the study group.

Part II: Corneal Vascularization vs Age, Sex, Refractive Error, Daily Wearing Time and Total Wearing Time

Age

Fig. 15 shows the age frequency distribution of the study group by number. Plotting corneal vascularization frequency as a percentage of each age group results in the histogram shown in Fig. 16 and summarized in Table 9. The sample sizes in the 0–9, 60–69 and 70–79 age groups are small (N<5), so vascularization frequencies for these age groups must be interpreted accordingly or disregarded altogether. Because the small observed frequencies in these age groups resulted in expected frequencies of less than 5, Chi-square analysis again could not be applied. Disregarding these age groups then and considering only the remaining age groups which were sufficiently large for statistical comparison, Chi-square analysis showed no significant difference in the frequency of vascularization as a function of age in the soft contact lens group.

The prevalence of vascularization for the control group is shown as a function

TABLE 6
Magnitude of Corneal Vascular Growth From Limbus
(Study Group N=280)

Distance (mm)	Males	Females	Totals
0.5	56 (20.0%)	120 (42.9%)	176 (62.9%)
1.0	32 (11.4%)	52 (18.6%)	84 (30.0%)
1.5	5 (1.8%)	6 (2.1%)	11 (3.9%)
2.0	3 (1.1%)	1 (0.4%)	4 (1.4%)
2.5	1 (0.4%)	3 (1.1%)	4 (1.4%)
3.0	0 (0.0%)	0 (0.0%)	0 (0.0%)
3.5	0 (0.0%)	0 (0.0%)	0 (0.0%)
4.0	0 (0.0%)	1 (0.4%)	1 (0.4%)
4.5	0 (0.0%)	0 (0.0%)	0 (0.0%)
	97 (34.6%)	183 (65.4%)	280 (100.0%)

TABLE 7
Magnitude Of Corneal Vascular Growth From Limbus
(Control Group N=18)

Distance (mm)	Males	Females	Totals
0.5	2 (11.1%)	0 (0.0%)	2 (11.1%)
1.0	3 (16.7%)	1 (5.6%)	4 (22.2%)
1.5	0 (0.0%)	2 (11.1%)	2 (11.1%)
2.0	1 (5.6%)	3 (16.7%)	4 (22.2%)
2.5	1 (5.6%)	2 (11.1%)	3 (16.7%)
3.0	0 (0.0%)	2 (11.1%)	2 (11.1%)
3.5	0 (0.0%)	0 (0.0%)	0 (0.0%)
4.0	0 (0.0%)	0 (0.0%)	0 (0.0%)
4.5	0 (0.0%)	1 (5.6%)	1 (5.6%)
	7 (38.9%)	11 (61.1%)	18 (100.0%)

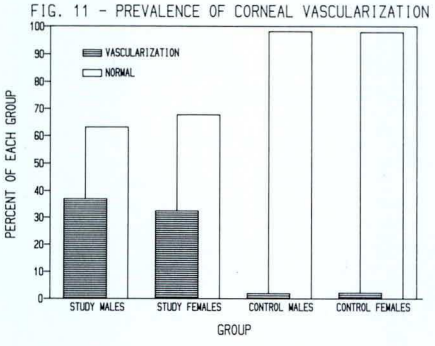

Figure 11
Prevalence of normal and vascularized corneas in the soft contact lens (study) group and the non-contact lens (control) group by sex.

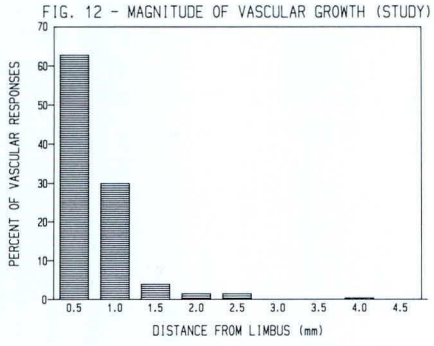

Figure 12
Extent of vessel penetration into the cornea from the edge of the translucent limbal zone for the soft contact lens (study) group.

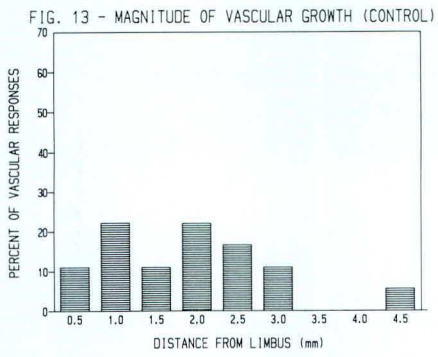

Figure 13
Extent of vessel penetration into the cornea from the edge of the translucent limbal zone for the non-contact lens (control) group.

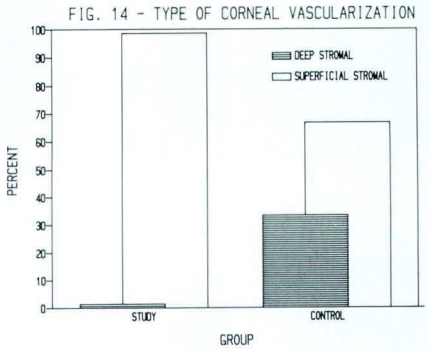

Figure 14
Prevalence of superficial and deep stromal corneal vascularization in the soft contact lens (study) group and the non-contact lens (control) group.

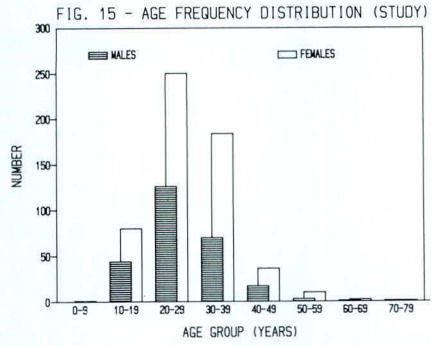

Figure 15
Number of patients in the soft contact lens (study) group by age and sex.

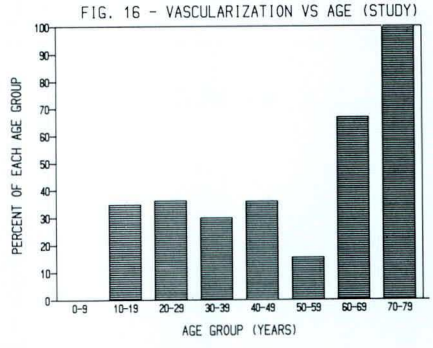

Figure 16
Prevalence of corneal vascularization in the soft contact lens (study) group by age.

TABLE 8
Classification Type Of Corneal Vascular Responses
(Study Group N=280) (Control Group N=18)

Group	Superficial Stromal	Deep Stromal
Study	276/280 (98.6%)	4/280 (1.4%)
Control	12/18 (66.7%)	6/18 (33.3%)

TABLE 9
Prevalence Of Vascularization For Each Age Group
(Study Group N=827)

Age Group	Males	Females	Totals
0-9	0/0 (0.0%)	0/1 (0.0%)	0/1 (0.0%)
10-19	13/44 (29.5%)	30/80 (37.5%)	43/124 (34.7%)
20-29	48/126 (38.1%)	88/251 (35.1%)	136/377 (36.1%)
30-39	28/70 (40.0%)	48/184 (26.1%)	76/254 (29.9%)
40-49	7/17 (41.2%)	12/36 (33.3%)	19/53 (53.8%)
50-59	0/3 (0.0%)	2/10 (20.0%)	2/13 (15.4%)
60-69	0/1 (0.0%)	2/2 (100.0%)	2/3 (66.7%)
70-79	1/1 (100.0%)	1/1 (100.0%)	2/2 (100.0%)
	97/262 (37.0%)	183/565 (32.4%)	280/827 (33.9%)

of age in Figs. 17–18 and Table 10. Once again, the small number of patients with vascularization in the control group precluded Chi-square analysis with the study group or between age groups within the control group itself. In the control group, the age groups 0–9 years and 70–79 years were comprised of small samples ($N < 10$), so vascularization frequencies for these age groups must also be interpreted with care or disregarded totally as with the study group. Visual inspection of Fig. 18, disregarding the age groups 0–9 years and 70–79 years, shows a slight increase in vascularization frequency with age in the control group, although whether this is statistically significant is uncertain. Since most cases of vascularization in the control group were due to acute causes, such as keratitis and ocular injuries which would be expected to occur randomly throughout life, one might expect to see fewer of these cases in younger patients, which indeed is the situation here.

Sex

The data shown in Figs. 10–11 and Table 5, when tested by Chi-square analysis, showed no significant difference at the 0.01 level in the prevalence of corneal vascularization for either males or females in the study group [$X^z=1.52$, df=1] or the control group [$X^z=0.00$, df=1].

Refractive Error

The distribution of refractive errors for the study group is shown in Fig. 19 and Table 11. The greatest number of refractive errors were in the −4.00 D to 0.00 D range (70.2%), followed by the −8.00 D to −4.00 D range (23.0%), for a cumulative frequency of 93.2% of refractive errors within the −8.00 D to 0.00 D range.

The distribution in Fig. 20 and Table 12, shows the prevalence of corneal vascularization as a function of refractive error. Chi-square tests of significance of each refractive error group with every other refractive error group did not produce valid results in every case because of expected frequencies less than 5. Of the data which could be compared however, it was discovered that patients wearing soft lenses in the power range of −8.00 D to −4.00 D had significantly more corneal vascularization at the 0.01 level than patients in the −4.00 D to 0.00 D range. No significant difference in corneal vascularization frequency was

found for patients in the −8.00 D to −4.00 D range with those in the 0.00 D to +4.00 D and +4.00 D to +8.00 D ranges.

Daily Wearing Time

Fig. 21 and Table 13 show the frequency distribution of daily wearing times for the study group. The majority of soft contact lens wearers (75.1%) were found to wear their lenses between 8 and 16 hours per day. There were 5.2% of patients who wore their soft lenses on an extended-wear basis. Fig. 22 and Table 14 show a smooth increase in frequency of vascularization as a function of daily wearing time until the 20–24 hour range at which point a slight drop in vascularization frequency is noted. Statistically, patients who wore their lenses 12–20 hours per day had significantly more corneal vascularization than patients who wore their lenses less than 12 hours per day ($p < 0.01$). Patients wearing soft lenses on an extended-wear basis had no significant difference in vascularization frequency from patients who wore daily-wear soft lenses 12 hours per day or less ($p < 0.01$). There was also no significant difference in vascularization frequency between groups who wore their lenses less than 12 hours per day ($p < 0.01$).

Total Wearing Time

Fig. 23 and Table 15 show the frequency distribution of total wearing times for the soft contact lens group. The majority (72.5%) of soft contact lens wearers had been wearing lenses five years or less. Only 27.5% of the study group had been wearing soft contact lenses more than five years.

Fig. 24 and Table 16 show vascularization frequency versus total soft contact lens wearing time in years. The prevalence of vascularization shown in Fig. 24 for the 11 and 12 year groups should be disregarded because of the small sample sizes ($N < 10$).

Chi-square analysis of each total wearing time period with every other total wearing time period established that patients who had been wearing soft contact lenses two years or less had significantly less corneal vascularization frequencies than those who had been wearing soft lenses three years or more ($p < 0.01$). After three years of soft contact lens wear, there was no significant increase in vascularization frequency as the total number of years of contact lens wear increased ($p < 0.01$).

TABLE 10
Prevalence Of Vascularization For Each Age Group
(Control Group N=900)

Age Group	Males	Females	Totals
0-9	0/1 (0.0%)	0/2 (0.0%)	0/3 (0.0%)
10-19	0/79 (0.0%)	0/109 (0.0%)	0/188 (0.0%)
20-29	0/63 (0.0%)	3/95 (3.2%)	3/158 (1.9%)
30-39	1/87 (1.1%)	4/144 (2.8%)	5/231 (2.2%)
40-49	2/89 (2.2%)	3/111 (2.7%)	5/200 (2.5%)
50-59	3/43 (7.0%)	0/45 (0.0%)	3/88 (3.4%)
60-69	1/9 (11.1%)	0/17 (0.0%)	1/26 (3.8%)
70-79	0/3 (0.0%)	1/3 (33.3%)	1/6 (16.7%)
	7/374 (1.9%)	11/526 (2.1%)	18/900 (2.0%)

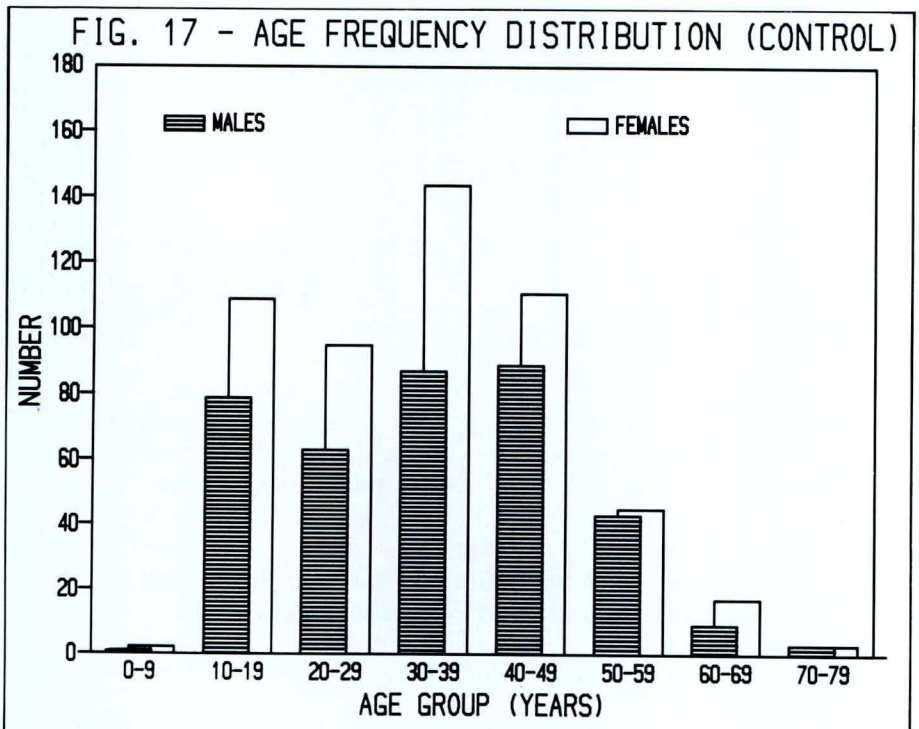

Figure 17
Number of patients in the non-contact lens (control) group by age and sex.

TABLE 11
Refractive Error Group Distribution
(Study Group N=732)

Rx Range (D)	Males	Females	Totals
−16−12	1 (0.1%)	2 (0.3%)	3 (0.4%)
−12−8	5 (0.7%)	10 (1.4%)	15 (2.0%)
−8−4	56 (7.7%)	112 (15.3%)	168 (23.0%)
−4−0	154 (21.0%)	360 (49.2%)	514 (70.2%)
0+4	5 (0.7%)	11 (1.5%)	16 (2.2%)
+4+8	4 (0.5%)	9 (1.2%)	13 (1.8%)
+8+12	2 (0.3%)	0 (0.0%)	2 (0.3%)
+12+16	0 (0.0%)	1 (0.1%)	1 (0.1%)
	227 (31.0%)	505 (69.0%)	732 (100.0%)

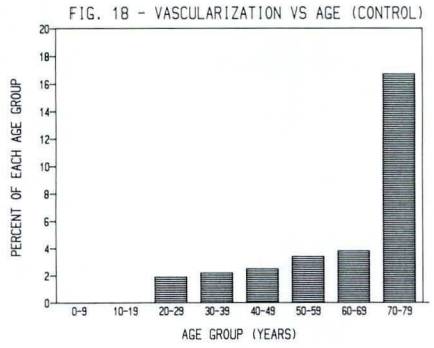

Figure 18
Prevalence of corneal vascularization in the non-contact lens (control) group by age.

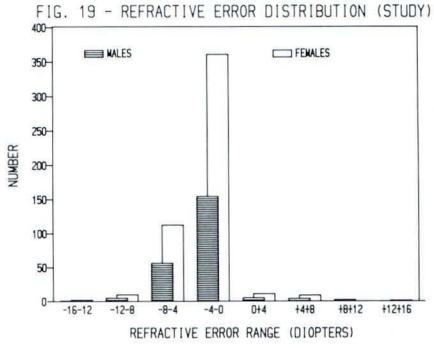

Figure 19
Number of patients in the soft contact lens (study) group by refractive error.

TABLE 12
Prevalence of Vascularization For Each Refractive Error Group (Study Group N=732)

Rx Group (D)	Males	Females	Totals
−16−12	0/1 (0.0%)	1/2 (50.0%)	1/3 (33.3%)
−12−8	3/5 (60.0%)	2/10 (20.0%)	5/15 (33.3%)
−8−4	28/56 (50.0%)	45/112 (40.2%)	73/168 (43.5%)
−4−0	47/154 (30.5%)	101/360 (28.1%)	148/514 (28.8%)
0+4	2/5 (40.0%)	1111 (9.1%)	3/16 (18.8%)
+4+8	2/4 (50.0%)	2/9 (22.2%)	4113 (30.8%)
+8+12	1/2 (50.0%)	0/0 (0.0%)	1/2 (50.0%)
+12+16	0/0 (0.0%)	1/1 (100.0%)	1/1 (100.0%)
	83/227 (36.6%)	153/505 (30.3%)	236/732 (32.2%)

TABLE 13
Daily Wearing Time Frequency Distribution (Study Group N=827)

D.W.T. (Hr)	Males	Females	Totals
0-4	10 (1.2%)	12 (1.5%)	22 (2.7%)
4-8	34 (4.1%)	77 (9.3%)	111 (13.4%)
8-12	75 (9.1%)	200 (24.2%)	275 (33.3%)
12-16	111 (13.4%)	235 (28.4%)	346 (41.8%)
16-20	20 (2.4%)	10 (1.2%)	30 (3.6%)
20-24	12 (1.5%)	31 (3.7%)	43 (5.2%)
	262 (31.7%)	565 (68.3%)	827 (100.0%)

Discussion

McMonnies et al[16] were concerned about the high frequency of limbal vascular engorgement which they found only in soft contact lens wearers. Their concern was that these patients may be predisposed, by virtue of their limbal vascular engorgement, to develop corneal vascularization at some later date. The results of this study would seem to support their concern.

It should again be emphasized that the criteria by which corneal vascularization was diagnosed in this study were those as discussed in Duke-Elder[18] and recommended by McMonnies et al[16]: the presence of blood vessels *outside the zone of normal limbal translucency* in clear corneal tissue. During biomicroscopy, this was best scrutinized by using high slit-lamp magnifications (25.6–40.0×), high intensity red-free illumination and a combination of direct and indirect retroillumination techniques.

Prevalence

We found that 33.9% of soft contact lens wearers had corneal vascularization compared to 2.0% of the control group. Moreover, 10/18 cases of vascularization in the control group were due to distinctly identifiable pathological causes: keratitis and ocular injuries. Hence only 8/900 cases (0.9%) of the entire control group were actually diagnosed as having corneal vascularization of unknown causes. By comparison, none of the soft contact lens wearers had known pre-existing identifiable pathological conditions which could have been expected to produce corneal vascularization. If one deducts the 0.9% of cases of corneal vascularization of unknown origin which were found in the control group from the 33.9% found in the soft contact lens group, the prevalence of soft contact lens related vascularization would then be 33.0%. The reason why corneal vascularization does not develop in all soft contact wearers is a question which remains to be answered. The answer may lie not only in how soft lenses alter normal corneal physiology but also how individual differences in patient corneal oxygen requirements influence the maintenance of corneal health.

Virtually all soft contact lens wearers with corneal vascularization were unaware of its presence. There were no subjective symptoms reported, except in

severe instances in which the vessels had advanced 4.5 mm from the limbus. The ocular symptoms then reported were: burning and itching, redness and blurred vision, as a result of the advanced stages of corneal edema, epithelial disturbance and limbal vascular injection, all of which were observed in these cases.

Magnitude

Corneal vascular responses of low magnitude occurred in far greater numbers in soft contact lens wearers than in non-contact lens wearers (Figs. 12 and 13). Note that the majority of soft contact lens related corneal vascular responses are within 0.5 mm of the limbus, followed by rapidly decreasing amounts in subsequent ranges, whereas in the control group there is an almost constant vascularization frequency as a function of vessel penetration distance from the limbus. In the control group, we know that 10/18 or 55.6% of the cases of corneal vascularization had causes of an acute nature: keratitis and ocular injuries, rather than slowly progressive conditions. Also, in every one of these cases the magnitude of the vascular response was 2.0 mm or greater. This is in direct contrast to the soft lens group which had the majority of cases of corneal vascularization magnitudes within 0.5 mm of the limbus, followed a smooth and rapid decrease in the number of cases as corneal vessel penetration distance increased from the limbus. This dissimilarity in the distributions of vascularization magnitudes attests to the slowly progressive, chronic nature of soft contact lens induced corneal vascularization.

When one considers that the oxygen transmissibility of soft contact lenses may be somewhat less than optimal to sustain normal corneal health, it is conceivable that certain patients' corneas are in a chronically oxygen-deficient state; enough so that low-grade edema may be present virtually all of the time. This chronic low-grade edema might not be sufficient to cause subjective symptoms in the early stages, so that over a period of time corneal vascularization could develop without the patient's awareness. This interpretation would correspond to the findings of this study: most soft lens patients with corneal vascularization were totally unaware of its presence.

In both the study and control groups, the maximum vessel penetration distance did not exceed 4.5 mm into the cornea. In the soft contact lens group, 96.8% of

TABLE 14
Prevalence of Vascularization For Each Daily Wearing Time Period (Study Group N=827)

D.W.T. (Hr)	Males	Females	Totals
0-4	0/10 (0.0%)	3/12 (25.0%)	3/22 (13.6%)
4-8	4/34 (11.8%)	14/77 (19.5%)	19/111 (17.1%)
8-12	23/75 (30.7%)	57/200 (28.5%)	80/275 (29.1%)
12-16	53/111 (47.7%)	95/235 (40.4%)	148/346 (42.8%)
16-20	11/20 (55.0%)	4/10 (40.0%)	15/30 (50.0%)
20-24	6/12 (50.0%)	9/31 (29.0%)	15/43 (34.9%)
	97/262 (37.0%)	182/565 (32.2%)	280/827 (33.9%)

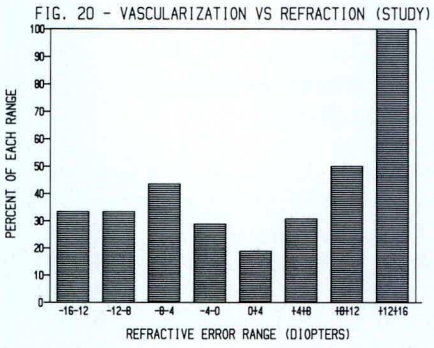

Figure 20
Prevalence of corneal vascularization in the soft contact lens (study) group by refractive error.

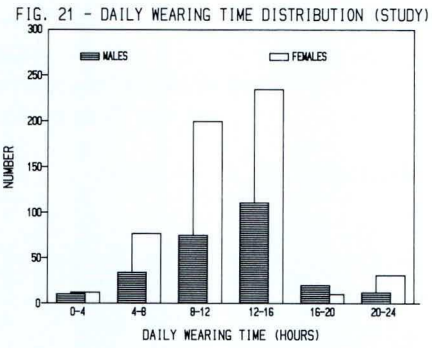

Figure 21
Number of patients in the soft contact lens (study) group by daily wearing time.

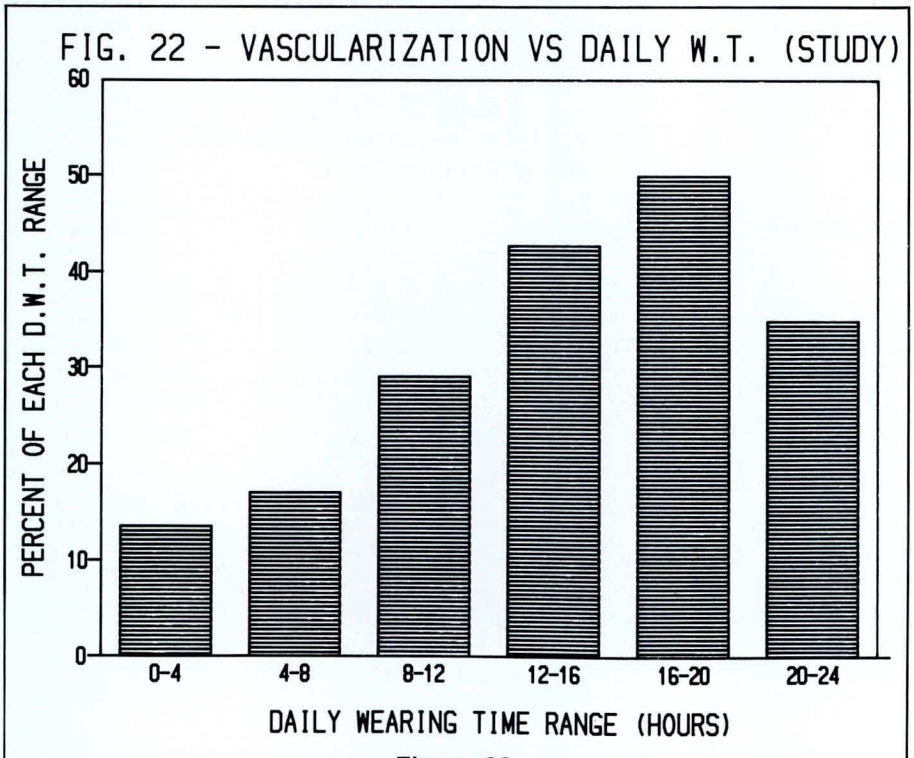

Figure 22
Prevalence of corneal vascularization in the soft contact lens (study) group by daily wearing time.

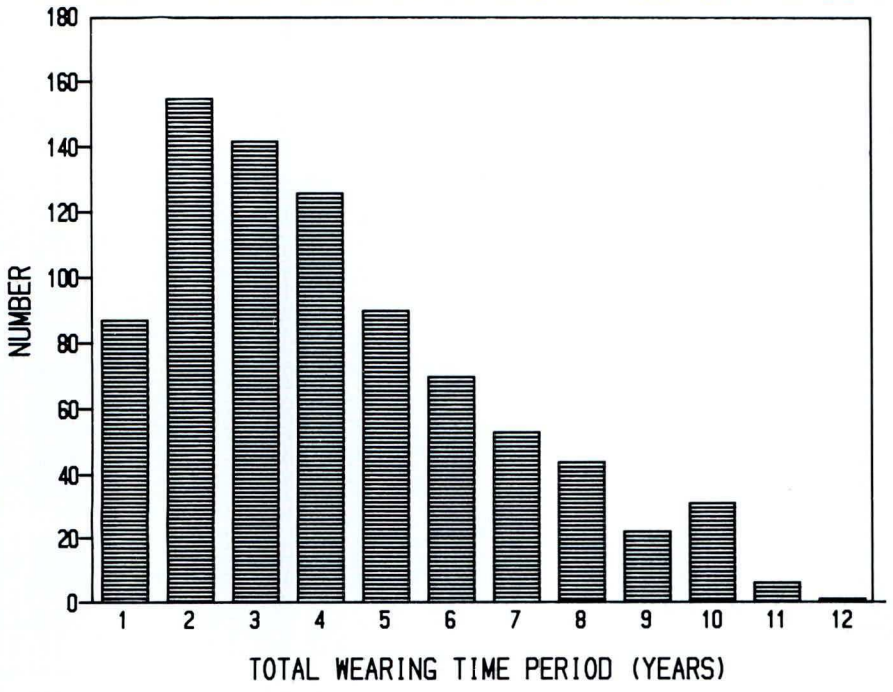

Figure 23
Number of patients in the soft contact lens (study) group by total wearing time.

TABLE 15
Total Wearing Time Frequency Distribution
(Study Group N=827)

T.W.T. (Yr)	Males	Females	Totals
1	33 (4.0%)	54 (6.5%)	87 (10.5%)
2	56 (6.8%)	99 (12.0%)	155 (18.7%)
3	53 (6.4%)	89 (10.8%)	142 (17.2%)
4	34 (4.1%)	92 (11.1%)	126 (15.2%)
5	34 (4.1%)	56 (6.8%)	90 (10.9%)
6	17 (2.1%)	53 (6.4%)	70 (8.5%)
7	17 (2.1%)	36 (4.4%)	53 (6.4%)
8	8 (1.0%)	36 (4.4%)	44 (5.3%)
9	3 (0.4%)	19 (2.3%)	22 (2.7%)
10	6 (0.7%)	25 (3.0%)	31 (3.7%)
11	1 (0.1%)	5 (0.6%)	6 (0.7%)
12	0 (0.0%)	1 (0.1%)	1 (0.1%)
	262 (31.7%)	565 (68.3%)	827 (100.0%)

the vascular responses were within 1.5 mm of the limbus. There was one case of a soft contact lens wearer displaying a severe vascular response in which vessels penetrated 4.5 mm into the cornea, although this represented only 0.4% of the study group. The fact that 96.8% of the vascular responses in the study group were limited to the peripheral 1.5 mm of the cornea, thus avoiding the central optic zone, explains why visual symptoms were reported only in the most advanced cases. By comparison, only 44.4% of vascular responses were 1.5 mm or less into the cornea in the control group, indicating the more serious nature of the conditions which caused the corneal vascularization in these cases.

Corneal edema and the formation and accumulation of vaso-stimulating substances such as lactic acid have been found to play an active role in corneal vascularization[14]. In soft contact lens wearers, it would appear that the effect of corneal edema and vaso-stimulating substances is most often limited to a peripheral annual zone extending inward about 1.5 to 2.0 mm from the limbus. In theory, this observation would concur with the fact that 93.6% of patients in the study group were myopes whose soft lenses would therefore be thicker peripherally than centrally, with correspondingly lower oxygen transmissibilities in the periphery. This could conceivably result in an annular area of localized edema and/or vaso-stimulating substance accumulation in the corneal periphery which could ultimately stimulate, while at the same time limit, corneal vascularization to this specific area.

Type

Another indicator of the fundamental difference in etiology of corneal vascularization in the study group from that of the control group is seen in Fig. 14. A much larger number of cases of superficial stromal corneal vascularization are observed in the study group compared to the control group. This could be explained by the fact that the conjunctival limbal circulation would be expected to be primarily involved in the vascular process in a group of soft contact lens wearers since soft lenses impinge physically upon the limbal vessels and corneal adnexa. However, as Tomlinson and Haas[15] reported, it is not the physical action of the contact lenses upon the limbal area which is the cause of vascular

injection and corneal vascularization, but rather corneal hypoxia which interferes with aerobic metabolism in this area. This would result in the development of corneal edema as well as the buildup of the byproducts of anaerobic metabolism such as lactic acid (a known vasostimulating substance), which are both required in the pathogenesis of corneal vascularization[14].

Age and Sex

The age and sex of patients were not found to be significant factors in the prevalence of corneal vascularization. This would indicate that there is no specific physical or physiological differences between sexes or between age groups which would predispose one particular group to corneal vascularization.

Refractive Error

Patients whose refractive errors were in the ranges from -8.00 to -4.00 D, 0.00 to +4.00 D and +4.00 to +8.00 D had corneal vascularization frequencies which were not significantly different from one another but *were* significantly higher than patients in the −4.00 to 0.00 D range. This could be explained if the oxygen transmissibility of soft lenses in the −4.00 to 0.00 D range was greater than that of lenses outside this range. We do not have specific data to confirm this, however it would seem to be a reasonable assumption since the average thickness of soft lenses in the −4.00 to 0.00 D range would be less than lenses outside this range. Oxygen transmissibility (Dk/L) is dependent upon Dk (oxygen permeability of the lens material) as well as L (average thickness of the lens material), so the greater the average thickness of a lens of a given water content, the less oxygen which is going to be available to the eye. Eyes wearing soft lenses of increasingly greater average thickness would therefore be expected to develop increasingly greater levels of corneal vascularization more often, as was in fact found.

Daily Wearing Time

Theory would predict that the longer the daily period of sustained corneal hypoxia due to soft contact lens wear, the greater the level of corneal edema and related corneal physiological changes; hence the greater the number of corneal vascular

TABLE 16
Prevalence of Vascularization For Each Total Wearing Time Period (Study Group N=827)

T.W.T. (Yr)	Males	Females	Totals
1	5/33 (15.2%)	11/54 (20.4%)	16/87 (18.4%)
2	14/56 (25.0%)	21/99 (21.2%)	35/155 (22.6%)
3	22/53 (41.4%)	29/89 (32.6%)	51/142 (35.9%)
4	14/34 (41.2%)	31/92 (33.7%)	45/126 (35.7%)
5	12/34 (35.3%)	16/56 (28.6%)	28/90 (31.1%)
6	9/17 (52.9%)	21/53 (39.6%)	30/70 (42.9%)
7	10/17 (58.8%)	16/36 (44.4%)	26/53 (49.1%)
8	7/8 (87.5%)	16/36 (44.4%)	23/44 (52.3%)
9	2/3 (66.7%)	6/19 (31.6%)	8/22 (36.4%)
10	2/6 (33.3%)	12/25 (48.0%)	14/31 (45.2%)
11	0/1 (0.0%)	3/5 (60.0%)	3/6 (50.0%)
12	0/0 (0.0%)	1/1 (100.0%)	1/1 (100.0%)
	97/262 (37.0%)	183/565 (32.4%)	280/827 (33.9%)

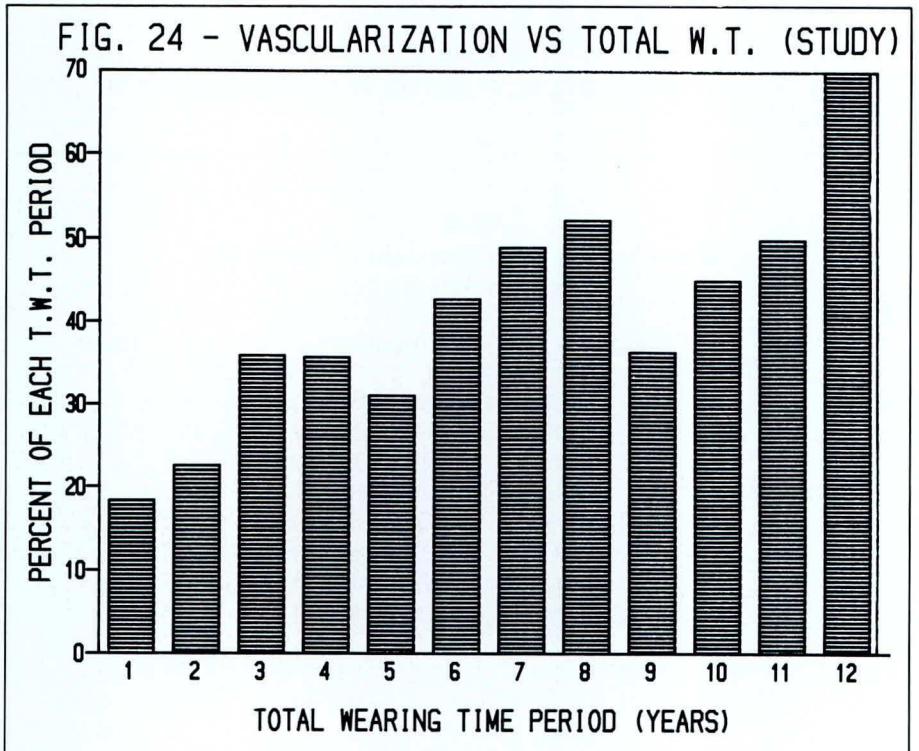

Figure 24
Prevalence of corneal vascularization in the soft contact lens (study) group by total wearing time.

responses. Increasing vascularization frequency as a function of daily wearing time is exactly what was found in this study, with one exception (Fig. 22). Note that as the daily contact lens wearing times increase, the frequency of corneal vascularization also increases until the 20–24 hour daily wearing time is reached, at which point a slight drop in vascularization frequency is noted. The drop in vascularization frequency at the 20–24 hour level could be explained by the fact that all of the patients of this wearing time group were extended-wear patients who were wearing soft lenses with greater oxygen transmissibility than the daily-wear lenses which the remainder of the study group were wearing.

Statistically, the frequency of corneal vascularization found in the extended-wear group was no different than that found in patients who wore daily-wear lenses 12 hours per day or less. Interestingly, both of these groups had significantly lower frequencies of corneal vascularization than patients wearing daily-wear lenses 12–20 hours per day. This does not imply that extended-wear lenses are not associated with significant levels of corneal vascularization: the frequency was 34.9% in the extended-wear group. Nor is the implication that daily-wear lenses worn less than 12 hours per day are without problems: the average vascularization frequency for the three daily-wearing time groups under 12 hours was 19.9%. It does imply, however, that patients who are wearing daily-wear lenses 12–20 hours per day are likely experiencing greater levels of corneal hypoxia and edema than those who are wearing daily-wear lenses less than 12 hours per day or patients who are wearing extended-wear lenses on an extended-wear basis.

Total Wearing Time

The prevalence of corneal vascularization found in patients during the first and second years of soft contact lens wear was not significantly different ($p < 0.01$), but both were significantly lower than that found in patients after three years of soft lens wear ($p < 0.01$). After three years, no statistically significant changes in the prevalence of corneal vascularization were found ($p < 0.01$). Thus corneal vascularization may take as long as three years to develop in soft contact lens wearers, indicating once again the slowly progressive nature of the condition.

Summary

1. Corneal vascularization was found in 33.9% of patients who had worn soft contact lenses for at least one year.
2. 96.8% of the vascular responses did not penetrate more than 1.5 mm into the cornea from the limbus.
3. Symptoms were almost never reported by patients who had soft contact lens related corneal vascularization.
4. Age and sex were not related to the prevalence of corneal vascularization in soft contact lens wearers.
5. Corneal vascularization was found in signficantly greater numbers in soft contact lens patients whose refractive errors were outside the range −4.00 to 0.00 D.
6. Patients who wore daily-wear soft lenses 12–20 hours per day had a significantly higher prevalence of corneal vascularization than patients who wore daily-wear lenses less than 12 hours per day, or patients who wore extended-wear soft lenses on an extended-wear basis.
7. Corneal vascularization increased over the first three years of soft contact lens wear after which time the prevalence remained constant.

Conclusions

Corneal vascularization is a relatively frequent occurrence in soft contact lens wearers. Although the condition itself does not cause serious visual problems, the fact that it occurs at all indicates a physiological intolerance to contact lenses which must be addressed. The fact that corneal vascularization is asymptomatic in the early stages confirms the need for monitoring the corneal health of soft contact lens wearers on a periodic and continuing basis.

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References

1. Dixon, J.M. and Lawaczeck, M.D. Corneal vascularization due to contact lenses. *Arch Ophthal* 1963; Vol 69: 72.
2. Mandelbaum, J. Corneal vascularization in aphakic eyes following the use of contact lenses — a report of two cases. *Arch Ophthal* 1964; Vol 71: 633.
3. Dixon, J.M. Ocular changes due to contact lenses. *Am J Ophthal* 1964; Vol 58: 424.
4. Dixon, W.S. and Bron, A.J. Fluorescein angiographic demonstration of corneal vascularization in contact lens wearers. *Am J Ophthal* 1973; Vol 75: 1010.
5. Dohlman, C.H. Boruchoff, S.A. and Mobilia, E.F. Complications in use of soft contact lenses in corneal disease. *Arch Ophthal* 1973; Vol. 90: 367.
6. Mosquera, J.M., Voss, E.H. and Moguilner, N.J. Corneal angiography and contact lenses. *Int Cont Lens Clin* 1974; Vol 1: 94.
7. Hewett, L. Clinical responsibility in fitting contact lenses. *Aust J Optom* 1974; Vol 57: 381.
8. Maino, J.H. and Carty, R.E. Neovascularization of the cornea secondary to contact lens wear. *Optom Monthly* 1979; Vol 70: 804.
9. Sendele, D. Superior limbic keratoconjunctivitis in contact lens wearers. *Ophthal* 1983; Vol 90: 616.
10. Bloomfield, S., Jacobiec, F. and Theodore, F. Contact lens induced keratopathy: A severe complication extending the spectrum of keratoconjunctivitis in contact lens wearers. *Ophthal* 1984; Vol 91: 290.
11. Abel, R., Shovlin, D. and DePaolis, M. A treatise on hydrophilic lens induced superior limbic keratoconjunctivitis. *Intl Cont Lens Clin* 1985; Vol. 12: 116.
12. Zantos, S.G. and Holden, B.A. Ocular changes associated with continuous wear of contact lenses. *Aust J Optom* 1978; Vol 61: 418.
13. Weinberg, R.J. Deep corneal vascularization caused by aphakic soft contact lens wear. *Am J Ophthal* 1977; Vol 83: 121.
14. Jantzi, J.D. A review of the pathogenesis of corneal vascularization. *Can J Optom* 1986; Vol. 48: 124.
15. Tomlinson, A., Haas, D.D. Changes in corneal thickness and circumcorneal vascularization with contact lens wear. *Intl Cont Lens Clin* 1980; Vol 7: 45.
16. McMonnies, C.W., Chapman-Davies, A. and Holden, B.A. The vascular response to contact lens wear. *Am J Optom and Physiol Opt* 1982; Vol 59: 795.
17. McMonnies, C.W. Contact lens induced corneal vascularization. *Intl Cont Lens Clin* 1983; Vol 10: 12.
18. Duke-Elder. System of Ophthalmology. Vol 8: 676.

Canadian Vision Standards

J.K. Hovis *

As primary vision care providers, optometrists are often asked whether a patient's vision meets the standard established for a particular occupation. In order to address these inquiries appropriately, optometrists must have knowledge of the various vision standards. This article provides a summary of federally-mandated vision requirements as a reference. However, because these standards are under continual revision, it will be necessary to update them from time to time.

In this summary, tests and testing procedures are not listed if they are similar to standard clinical procedures. With respect to colour vision evaluation, patients *are not* allowed to use any coloured filter aids during testing to improve their colour perception. The use of a pseudoisochromatic plate test that is not one of the specified tests is usually a valid procedure, because individual test validity is high and inter-test correlations between many plate tests is quite good.[1] That is, a person who passes (or fails) one test has a high probability of passing (or failing) another test. Nevertheless, patients who "just-pass" or "just-fail" a nonspecified colour vision test should have their colour vision reassessed with the specified test. Patients who pass or fail the colour vision test in a vision screening machine should also have their colour perception reassessed, because many of these tests have a very low validity.[1] Several agencies require further testing with the Royal Canadian Navy (RCN) Lantern Test to determine if a colour defective person can safely perform his/her duties. Although this Lantern Test is no longer manufactured, a working model may be available at a Canadian Forces base or a rail company's regional office.

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School of Optometry
University of Waterloo

Vision Requirements for Engagement in the Royal Canadian Mounted Police[2]

Uncorrected visual acuity: 6/12 in better eye, 6/30 in other eye; or 6/18 in each eye.
Corrected visual acuity: 6/6 in better eye, 6/9 in other eye; or 6/6 in each eye.
Colour vision: Pass the 38 plate edition of the Ishihara Colour Vision Test (4 or less errors on plates 1 to 21); or if s/he fails, then the candidate must pass the Farnsworth-Munsell Panel D-15 Test (less than 2 transpositions that parallel an error axis).
Binocular vision: No strabismus or history of diplopia.
Visual fields: Binocular visual field extending at least 120° in the horizontal meridian.

Vision Standards for Merchant Marine Master and Mate Certificates[3]

Uncorrected visual acuity: 6/60 in each eye.

TABLE 1
Visual Acuity Grades for the Canadian Forces*.

Acuity Grade	Uncorrected Acuity		Corrected Acuity	
	Better Eye	Other Eye	Better Eye	Other Eye
V1	6/6	6/9	N/A	N/A
V2	6/18 or 6/12	6/18 or 6/30	6/6	6/6
V3	6/120	6/120	6/9	6/9
V4	N/A	N/A	6/9	6/120
	(As long as refractive error does not exceed ±7.00D in any meridian in the better eye)			
V5	Reserved for serving personnel with corrected acuity worse than 6/9, but, in the opinion of an ophthalmologist, can perform their duties satisfactorily.			
V6	Assigned to candidates who do not meet V4.			

*Refers to eyes that are unaltered by orthokeratology, and, presumably, radial keratotomy.

TABLE 3
Visual acuity and cycloplegic refraction standards for entry into Canadian Forces aircrew personnel.

	Pilot and Tactical Helicopter Observer				Search and Rescue Specialist				Other Aircrew			
	Better Eye		Other Eye		Better Eye		Other Eye		Better Eye		Other Eye	
	Distance	Near	Distance	Near	Distance	Near	Distance	Near	Distance	Near	Distance	Near
Uncorrected Visual Acuity	6/6	.65M (N5)	6/9	.75M (N6)	6/12	1.0M (N8)	6/30	1.3M (N12)	6/120	N/A	6/120	N/A
					6/18	or 1.2M (10)	6/18	1.2M				
Corrected Visual Acuity	N/A		N/A		6/6	.65M (N5)	6/9	.75M (N6)	6/6	.65M (N5)	6/9	.75M (N6)
Cycloplegic Refraction												
Sphere Power	Between −0.25 and +2.50D in either eye				N/A				Between −2.00D and +3.50D in either eye			
Cylinder Power	Not more than 0.75D in either eye				N/A				Not more than 1.25D in either eye			

TABLE 2
Colour Vision Grades for the Canadian Forces

Colour Vision Grade	Test Results
CV1	Passes Pseudoisochromatic Plate Test (this is either the Ishihara or AO Pseudoisochromatic Plates)
CV2	Fails the Plate Test, but passes the RCN Lantern Test
CV3	Fails the Plate and Lantern Tests

TABLE 4
Visual acuity and cycloplegic refraction standards for experienced aircrew in the Canadian Forces.

	Experienced Pilot		Other Experienced Aircrew	
	Better Eye	Other Eye	Better Eye	Other Eye
Uncorrected Visual Acuity	6/12	6/30	6/120	6/120
	or 6/18	6/18		
Corrected Visual Acuity	6/6	6/9	6/6	6/9
Cycloplegic Refraction				
Sphere Power	Between −1.00D and +3.50D		Between −2.00D and +3.50D	
Cylinder Power	Not more than 1.25D		Not more than 1.25D	

Corrected visual acuity: 6/9 in each eye, or if person began duty before June 1, 1973, then the standard is 6/9 with both eyes together.

Colour vision: The passing criteria for the three editions of the Ishihara Colour Vision Test are as follows:

1) 3 or less errors on plates 1 to 21 of the 38 plates edition,
2) 2 or less errors on plates 1 to 15 of the 24 plates edition,
3) 1 error on plates 1 to 8 of the 16 plates edition.

For candidates who fail the Ishihara Test, their colour vision can be assessed by RCN Lantern Test. If s/he fails the Lantern Test, then the candidate is rejected.

Vision Standards for the Canadian Forces[4]

Minimum visual acuity and colour vision requirements for various trades and assignments in the Canadian Forces are specified by numerical grades. Because there are over one hundred different positions with vision requirements, this paper only defines the visual acuity and colour vision grades. These are listed in Tables 1 and 2. Information concerning a specific assignment's standards can be obtained from a local recruiter. Aircrew candidates, however, must meet additional visual requirements and these are presented separately.

The vision requirements for any assignment or trade may be relaxed on an

TABLE 5
Vision standards for railway workers.

		Entrance into service	Promotion	Re-examination
A (Engineers, Motormen, Firemen, Diesel Helpers in Road Service); B (Engineers, Motormen, Diesel Helpers in Yard, Outside Hustler); and C (Brakemen, Yard Helpers, Switch Tenders, Operators, Towermen)	Uncorrected Visual Acuity	6/9 in better eye	6/9 in better eye	6/9 in better eye
		6/12 in other eye	6/15 in other eye	6/15 in other eye (Class B 6/9 in one eye regardless of other eye, Class C 6/6 in one eye regardless of other eye)
	Corrected Visual Acuity	6/6 in each eye	Same as uncorrected	Same as uncorrected
	Colour Vision	Normal	Normal	Normal
	Visual Fields	Normal	Normal (N/A for Class C)	Normal (N/A for Class C)
D (Conductors, Yardmasters, Yard Foremen, Train Baggagemen)	Uncorrected Visual Acuity	6/9 in better eye	6/9 in better eye	6/12 in better eye and 6/15 in other eye, or 6/9 in better eye
		6/2 in other eye	6/15 in other eye	and 6/21 in other eye, or 6/6 regardless of vision in other eye
	Corrected Visual Acuity	6/6 in each eye	6/6 in better eye, or meets uncorrected standards	Same as uncorrected
	Colour Vision	Normal	Normal	Normal
	Visual Fields	Normal	N/A	N/A
E (Partial list includes: Dispatchers, Station Agents, Signalmen, Welders)	Uncorrected Visual Acuity	6/9 in better eye	N/A	6/12 in better eye and 6/21 in other eye, or 6/9 in one eye regardless of vision in other eye
		6/15 in other eye		
	Corrected Visual Acuity	Same as uncorrected	N/A	Same as uncorrected
	Colour Vision	Normal	N/A	Normal
	Visual Fields	Normal	N/A	N/A
F (Flagmen, Watchmen, Gatemen)	Uncorrected Visual Acuity	6/12 in each eye	N/A	6/15 in one eye and 6/21 in other eye, or 6/9 in one eye regardless of vision in other eye
	Corrected Visual Acuity	Same as uncorrected	N/A	Same as uncorrected
	Colour Vision	Normal	N/A	Normal
	Visual Fields	Normal	N/A	N/A
G (Helpers operating snow plow or other equipment moving on truck coupled ahead of locomotive)		Same standards as for Class A re-examination	N/A	Same standards as for Class A re-examination

individual basis if the candidate has experience in that particular area.

Vision Standards for Aircrew Personnel Within the Canadian Forces[4]

Visual acuity: Acuity and cycloplegic refraction standards for entry level and experienced aircrew are listed in Tables 3 and 4.

Colour vision: The minimum grade is CV2.

Binocular vision: No diplopia or history of diplopia. No strabismus. Horizontal phorias should be greater than 6^{Δ}, and vertical phorias should not be greater than 1^{Δ}. Individuals with larger phorias will be considered eligible for aircrew duty depending upon experience, stereopsis, refusion speed, and refractive error.

Visual fields, slit lamp examination, and intraocular pressures must be within normal limits.

Vision Standards for Railway Workers[5,6]

Railway positions that have vision standards fall into one of seven classes. The seven classes and their visual requirements are outlined in Table 5. In addition to these requirements, all seven classes must have a minimum near visual acuity of .75M (corrected or uncorrected), and normal, single binocular vision (i.e., no strabismus or diplopia).

Colour vision can be assessed with either the Ishihara or AO Pseudoisochromatic Plates Colour Vision Tests. Errors on 28% or more of the screening plates constitutes a failure. A Lantern Test or Colour Threshold Tester (no longer commercially available) can be used to determine if a person who fails the plate test can accurately identify coloured signal lights. Persons who pass the secondary tests may be assigned to the position depending upon a committee's recommendations.

Visual fields are assessed by the confrontation method. Normal limits are

defined at 90° temporally, 60° nasally, 55° superiorly, and 70° inferiorly.

All employees are required to wear spectacles to correct their vision while working. However, contact lenses may be worn if the employee has undergone cataract surgery, cannot obtain the standard acuity by any other means, or has demonstrated that contact lenses are well tolerated when worn during work.

Vision Standards for Civil Aviation[7]

Transport Canada's rules and regulations were outlined in a 1976 issue of the Canadian Journal of Optometry[8]. Many amendments have since been added, and so the following updates the previously published requirements.

The number of licensing categories has recently increased from three to four. Category 1 applies to airline and commercial pilots; Category 2 applies to flight navigators, flight engineers, and air traffic controllers; Category 3 applies to private, glider, balloon, gyroplane, commercial gyroplane (in flight instruction), and all of the corresponding student pilots; and Category 4 applies to ultra-light aircraft pilots and student pilots. Category 4 has only two standards: distance visual acuity of at least 6/9 (corrected or uncorrected) in the better eye and normal visual fields.

The more stringent requirements for categories 1 to 3 are as follows:

Ocular health requirements for categories 1 to 3: There should be no active ocular pathology that could interfere with the applicant's performance.

Ocular muscle balance standards for categories 1 to 3: Lateral phoria limit (no strabismus) is 6^{Δ} and the vertical phoria limit is 1^{Δ}. Applicants who do not meet this standard will be referred to an ophthalmologist for further evaluation. The applicants may be licensed provided that diplopia is unlikely.

Colour vision requirements for categories 1 to 3: Colour perception is classified as normal if a patient passes any edition of the Ishihara Colour Vision Test, the AO Standard Pseudoisochromatic Plates, or the AOHRR Colour Vision Test. Minimum passing criterion for the Ishihara and AO Standard Tests is 83% correct on the screening plates. The passing criterion for the OAHRR test is 100% correct on the first six screening plates. Applicants who fail any one of these tests are administered a practical colour perception test to determine if their colour vision is adequate for performance of their duties. This test consists of identifying the colours of airport signal lights while in flight.

Commercial or private pilot license applicants who fail both the pseudoisochromatic and practical colour vision tests can be issued a license for daylight flying only, and they must have a 2-way radio in the plane for use at controlled airports.

Visual acuity standards for categories 1 to 3: Minimum corrected or uncorrected acuity is 6/9 in each eye. However, there are allowances for applicants who meet the 6/9 criterion with only one eye outlined at the end of this section. If correcting lenses are worn, then the applicant must have a spare pair of spectacles for immediate use when performing his/her duties. Contact lenses can be used as correcting lenses if vision is stable and the lenses are comfortable after a six month trial period. Contact lens wearers are required to have a pair of spectacles available for immediate use if the lenses are removed or become dislodged. The spectacle correction must allow them to meet the acuity standards. For lens wearers with appreciable spectacle blur, a third pair of spectacles may be required so that they can meet the standards immediately after lens removal and after the eye has stabilized.

Prescription sunglasses are not allowed as the second pair of correcting lenses during night flight.

Although the applicant is allowed to have correcting lenses in order to meet the distance acuity requrement, the lenses spherical equivalent power cannot be greater than ± 3.00 for categories 1 and 2 and ± 5.00D for category 3. Exceptions to the lens power requirements are possible for categories 2 and 3 applicants in accordance with medical opinion.

In addition to distance acuity standards, applicants in all three categories are required to have near visual acuities (corrected or uncorrected) of .65M (N5) at 40 cm and 1.60M (N14) at 100 cm. If a correction is needed to meet these standards, then the lens design must allow the applicant to meet the distance requirement without removing the lenses.

Allowances for monocular applicants and persons with substandard vision in one eye: Monocular applicants are persons whose visual acuity in one eye cannot be corrected to at least 6/60. These people may be granted a license if the uncorrected acuity in the better eye is 6/60 and can be corrected to a minimum of 6/9 with an equivalent sphere power of no greater than ± 3.00D. The function of the better eye must be normal in all other respects, and the applicant must pass a flight test. An optometrist's or ophthalmologist's report is required for each revalidation of the license.

Applicants with substandard vision in one eye are persons whose corrected acuity in one eye is between 6/9 and 6/60. For these applicants, the visual acuity, correcting lens power, and colour vision of the better eye must meet the appropriate standards, and medical opinion or a flight test indicates that the visual defect is unlikely to interfere with the applicants performance. Annual reports are required if the condition is progressive.

Acknowledgement

I thank Dr. William Bobier for comments on an early version of the manuscript.

References

1. Procedures for Testing Colour Vision, Report of Working Group 41. Committee on Vision, Assembly of Behavioral and Social Sciences, National Research Council. Washington, DC. National Academy Press, 1981.
2. MacDougall, J.A. Health Service Officer, RCMP "O" Headquarters, personal communication, January 1987.
3. Ghuman, T. Marine Surveyor, Ottawa, personal communication, October 1986.
4. Medical Standards for Canadian Forces, Government Publication A-MD-154-000/FP-000, 1979.
5. National Transportation Act of 1977, Chapter 1173, Railway Act General Order Nos. 0–9.
6. Canadian Transport Commission 1985–3 Rail, Canada Gazette, Part II, Vol. 119.
7. Transport Canada, Air, Personnel Licensing Handbook, Medical Requirements, Vol. 3, 1983, amendments from 1984 to 1986.
8. Schaefer, D.N. Visual requirements for civil aviation personnel licensing. *Can. J. Opt.* 38, 98–102, 1976.

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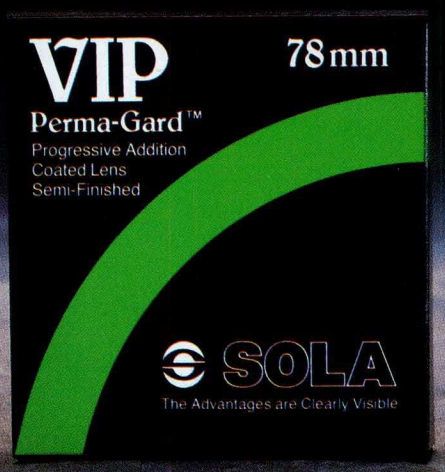

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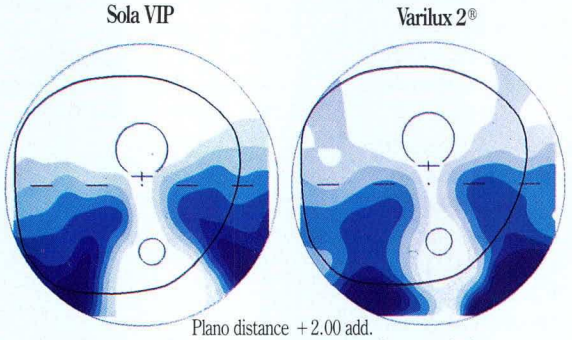

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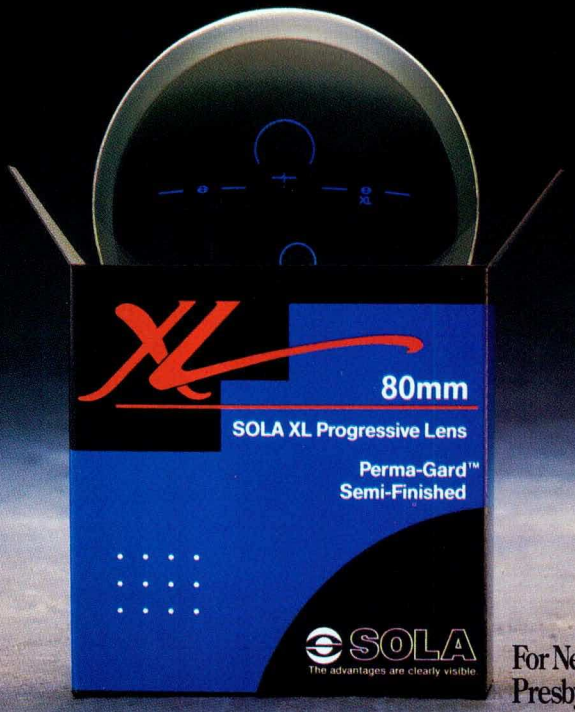

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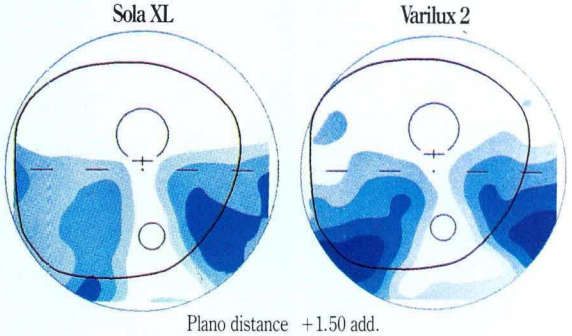

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The Canadian Association of Optometrists Gallery of Presidents

On the following two pages, CAO is pleased to publish its just completed Gallery of Presidents.

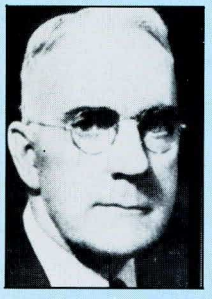
Beginning under Dr. H.A. McClung in 1941, the idea of a national optometric Association was shaped slowly through four Presidents until CAO was enacted by an Act of Parliament which received Royal assent in 1948. The Association at the time was under the Presidency of Dr. E. Boyaner.

Assembling the portraits that make up the completed Gallery was also a lengthy process. Initially requested in late 1984, the photos came in one by one until the deadlines imposed by the 1985 Biennial Congress caused us to unveil an incomplete Gallery at the 19th Biennial Congress. Although only three Presidential photos were missing at that time, they proved to be the most difficult to locate. Through the next two years, the search continued sporadically, with not a few frustrations and dead ends along the way. Finally, with deadlines for our 20th Biennial Congress pressing, the last portrait — of Dr. D.A. Maguire — was obtained on loan from the Museum of Canadian Optometry through the courtesy and generosity of Dr. E.J. (Ted) Fisher, who threatened dire reprisals if the original publication in which the photo was printed were to be waylaid in the Waterloo to Ottawa round trip.

It was with a great deal of pleasure, therefore, that we were able to present the complete portfolio at the Saint John Congress.

Throughout the process, the comments accompanying the photos loaned to us made the process of gathering them less of an ordeal. "Reprints are beyond price", advised Dr. Ivan McNabb and Dr. Harry Perrin noted that, "I look much better now than I did when this was taken."

The original Gallery now has a home in the CAO office conference room. Copies were also sent to each of the past national Association Presidents and it is included here for the enjoyment and memories of all those optometrists who have contributed to the growth and development of the Canadian Association of Optometrists.



Dr. H. McClung
1941–1943

Dr. A. Mignot
1943–1945

Dr. D. Maguire
1945–1946

Dr. F. Nuttall
1946–1948

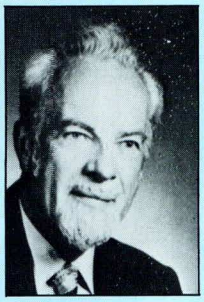
Dr. E. Boyaner
1948–1950

Dr. H. Perrin
1950–1951

Dr. H. Nowlan
1951–1952

Dr. J.J. Mulrooney
1952–1953

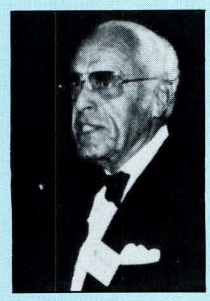
Dr. C.A. Palmer
1953–1955



Dr. W.M. Lyle
1955–1957

Dr. H.A. Coape-Arnold
1957–1959

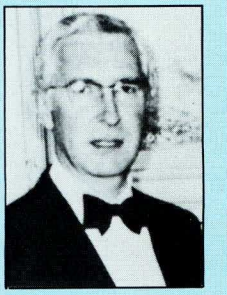
Dr. D.L. Francis
1959–1961



Dr. E.M. Finkleman
1961–1963

Dr. D.R. Price
1963–1965

Dr. E.N. Rea
1965–1967



Dr. H.D. Mackenzie
1967–1969

Dr. E.J. Spearman
1969–1971

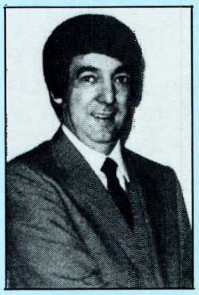
Dr. R.W. Macpherson
1971–1973

Dr. I.J. McNabb
1973–1975

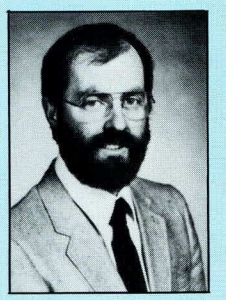
Dr. G.C. Lecker
1975–1977

Dr. R. Brown
1977–1979

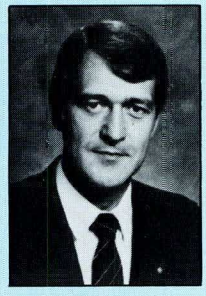
Dr. J.F. Huber
1979–1980



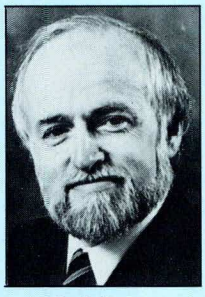
Dr. H.L. Landry
1980–1981



Dr. J.A.R. MacDuff
1981–1982

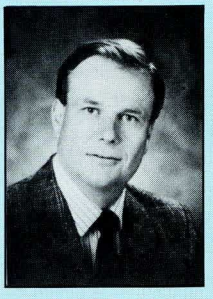


Dr. R.C. des Groseilliers
1982–1984



Dr. R. Rosere
1984–1985

Dr. B.N. Rosner
1985–1986



Dr. S.D. Brisbin
1986–1988

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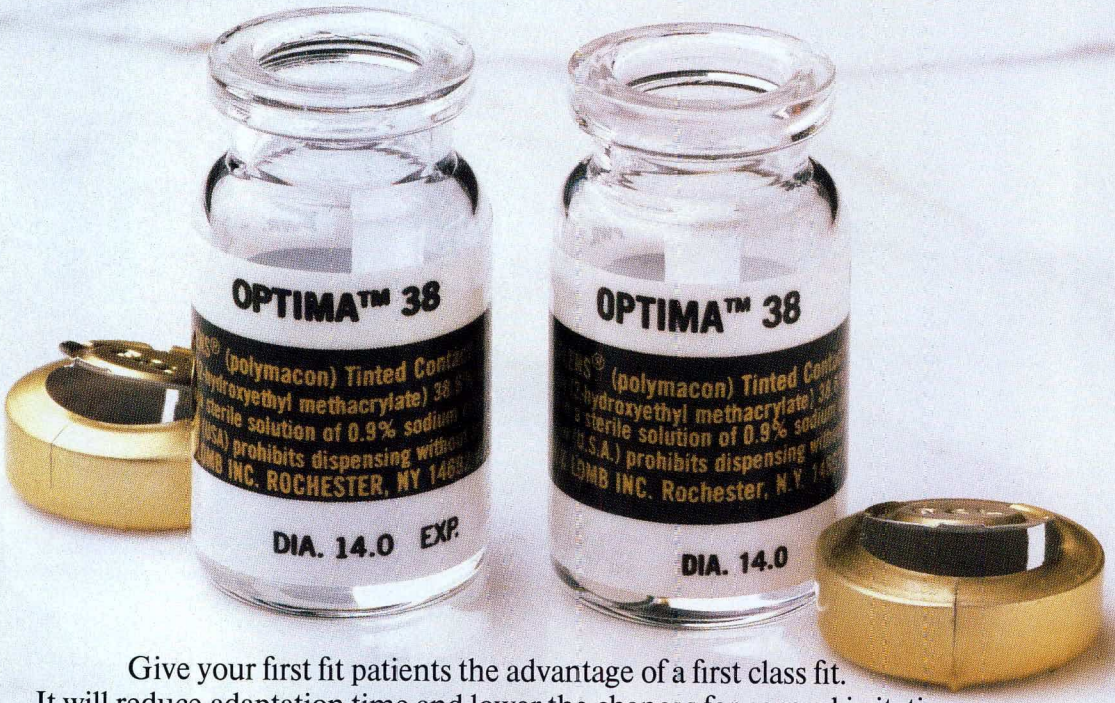

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The Development of β-Adrenergic Blocking Drugs for Management of Primary Open-Angle Glaucoma

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In the last few years, there has been a marked increase in the number of drugs available for the management of open-angle glaucoma. The development of these newer ophthalmic drugs has been prompted by a desire to make available, to the public, products that display maximum efficacy (with respect to the glaucoma) but minimal side effects on the body generally or on the health of the eye or on vision. The major development has been in a set of drugs active on β-adrenergic receptors (the "β-blockers"). While many of these newer drugs are, as yet, available only in Europe or Japan, in 1986, three new ophthalmic β-blockers became available in the English-speaking world.[1] It is our intent in this article to review why there has been this major expansion in the number of drugs available to manage glaucoma or ocular hypertension — an expansion that comes from many years experience with 8 different drugs worldwide.[a] With the increasing availability of these glaucoma medications to the optometrist in the USA[2], it is recognized that the optometrist in Canada will have been exposed to reports of the use of timolol, levobunolol and betaxolol by colleagues.[3] Indeed, the optometric community in the USA is continuing to lobby to make therapeutic drugs available to all optometrists.[4]

Drugs in the beta blocker classification are also widely administered as oral drugs to manage cardiac arrhythmia, (and associated angina or systemic hypertension) and sometimes migraine. Orally administered β-blockers can be expected to exert some action on the eye. For example, small ocular hypotensive effects ($\leq$ 10% change in IOP) can occur especially shortly after these drugs are taken. Although extensive studies have been conducted on the ocular hypotensive action of systemically administered β-blockers, this mode of administration of β-blockers has not been adopted in general practice as a means to manage glaucoma. These effects will therefore not be discussed further. Our review will concentrate on the use of β-blockers as eyedrops in Canada, the USA, Europe and Japan.

For those not familiar with β-blockers, a few words on terminology seem appropriate. β-blockers have been shown to bind to β-adrenergic receptors but do not activate them; instead these drugs deny the normal neurotransmitter (norepinephrine) access to beta adrenergic receptors.[5]. Drugs in this class may be also referred to as sympatholytic drugs or β-adrenoceptor antagonists. β_1 receptors are located in the heart and in the ciliary body particularly in the walls of blood vessels and some on muscle cells. Stimulating β_1 sites increases heart rate[6] and indirectly increases aqueous humor production. Blocking β_1 sites results in decreased heart rate and force of cardiac contraction,[5] and decreased aqueous production while having little effect on the pulmonary system. β_2 receptors are associated with smooth muscles in the walls of blood vessels and the lungs. Thus, stimulating β_2 receptors causes bronchodilatation, vasodilatation (or vasoconstriction depending on the site in the body) and acts on the ciliary body to increase aqueous production. There are many β_2 sites in the iris and ciliary body,[7] and in the trabecular meshwork vasculature. Stimulating β_2 sites in the trabecular region appears to facilitate escape of aqueous from the eye. Blocking β_2 sites causes the bronchi to constrict and causes some reduction in aqueous production while having little effect on heart rate.[5] Thus a β_2-blocker (or a drug with combined β_1, β_2- blocking activity) can exacerbate symptoms in asthmatics (even precipitating an asthmatic attack due to acute broncho-constriction). A β_1-blocker (or a drug with combined β_1,β_2-blocking activity) can precipitate bradycardia (slowing of heart rate below normal levels), exacerbate cardiac arrhythmias in a few patients and lower blood pressure below normal levels. Attention to these vital signs in a patient constitutes one aspect of patient management in the selection of the appropriate ophthalmic β-blocker although the clinical relevance of mean heart rate reductions of 5 to 10 bpm (4% to 8%) is questionable.

As indicated above, these β-blocking drugs rarely show absolute selectivity between β_1 or β_2 sites. Thus, these β-blockers can act by:

- blocking either β_1 or β_2 receptors. Those which preferentially block β_1 sites are called cardioselective (because they will not affect the pulmonary system at normal clinical doses).

a. Every effect has been made to provide correct information on drug availability, indications and contraindications, limits of use and accumulated reports of side effects. Sources of information include the articles listed below and the following pharmaceutical directories — Compendium of Pharmaceuticals and Specialities (Canada), 1987 Edition; Physicians Desk Reference — Ophthalmology (USA), 1987 Edition; Rote Liste (West Germany), 1987 Edition; British National Formulary (UK), 1987 Edition.

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- blocking both β_1 and β_2 receptors, these are called non-selective blockers.
- from an ocular perspective, two other factors need to be considered since some of these drugs have ***membrane stabilizing activity***. Repeated instillation of a drug which has this action can result in corneal anesthesia in some individuals and eventually punctate keratitis or general lid irritation. Some β-blockers have ***intrinsic sympathomimetic activity***. They have some ability to stimulate one or more types of adrenergic receptor (i.e. mimic the action of norepinephrine or epinephrine).

These functions are not always rigidly distinct, for example a drug may mostly block β_1 sites but if the dose is sufficient it will also block β_2 sites. At certain doses some β-blockers (depending on their potency) can show membrane stabilizing and/or intrinsic sympathomimetic activity.

A large number of β-blockers have been investigated to see if an eyedrop preparation of the drug will lower intraocular pressure.[5-9] (Table 1).

We will review several of these β-blockers in alphabetical order. For all of these drugs, the efficacy (i.e. the magnitude and time-base of the clinical effect) of any one drug (used in the form of eyedrops) has frequently been found to be enhanced by the use of a second topical ocular hypotensive drug (e.g. use of two ophthalmic β-blockers simultaneously or the concurrent use of epinephrine eyedrops or pilocarpine eyedrops). Orally-administered anti-glaucoma drugs (e.g. the carbonic anhydrase inhibitors such as acetazolamide) have also been shown to add to the ocular hypotensive action of a topical ophthalmic β-blocker. However, for the most part, reports of synergistic effects, although of academic interest, will not be commented upon further. In the clinical setting, such combined therapy is generally used only in patients who fail to respond adequately to single drug use or during changeover from one set of eyedrops to another. Two effective "combination" drugs have enjoyed considerable use for many years (epinephrine-pilocarpine combinations in Canada and USA and epinephrine-guanethidine combinations in Europe). Other anti-glaucoma combinations which are available in some countries include, pilocarpine + physostigmine, pilocarpine + neostigmine, pilocarpine + dipivefrin, guanethidine + dipivefrin, pilocarpine + phenylephrine, pilocarpine + metipranolol and pilocarpine + 3',4'-dihydroxy-2 methylaminoacetophenon.

TABLE 1
Beta adrenoreceptor blocking agents

	Division I non-cardioselective	Division II cardioselective
Group 1 have MSA and ISA	alprenolol bunolol carteolol oxprenolol pindolol [b]	acebutolol
Group 2 have MSA but no ISA	bupranolol labetalol [a] levobunolol [b] metipranolol [b] propranolol	
Group 3 have ISA but no MSA		practolol [c]
Group 4 have no MSA and no ISA	befunolol nadolol sotalol timolol [d]	atenolol [c] betaxolol [d] metoprolol [d] tolamolol [d]

MSA = membrane stabilizing activity, a quinidine-like effect, local anesthesia, eventually punctate keratitis
ISA = intrinsic sympathomimetic activity, may be desirable, hastens drug action

a - also blocks α_1 adrenoreceptor sites
b - has been said to have no MSA
c - has some ability to block β_2 sites as well
d - may have some MSA

Atenolol

Atenolol is a cardioselective β_1-blocker which may be employed to treat high blood pressure. It is not currently used in glaucoma therapy in Canada or USA although it underwent extensive trials in the late 1970s in a variety of topical formulations. It has no membrane stabilizing activity and almost no intrinsic sympathomimetic activity.[9] Topical atenolol 4% was found to have only small effects on blood pressure and heart rate.[10,11] Atenolol eyedrops (1% to 4%) produced a dose-dependent fall in IOP of 1 to 4 mmHg[10] or 4.9 to 6.3 mmHg[11,12] with a single instillation. Single drops of atenolol 4% were reported to lower IOP by an average of 5.6 mmHg (range 3.2 to 13.2 mm)[13] and two drops lowered IOP by 8 to 10 mmHg.[14] Similar results were also observed over one week with instillation twice a day for patients with initial pressures $\leq$ 30 mmHg[10]. However, atenolol 4% eyedrops, twice a day, appear to be unable to hold pressures down for more than a week or two especially if starting pressures are $\geq$ 30 mmHg.[15,16] A subsequent trial of atenolol 4% eyedrops (single drops) on normal healthy subjects indicated that the hypotensive effects of atenolol would only be observed in ocular hypertensive and/or glaucomatous eyes since the trial showed only 1 to 1.5 mm reduction in IOP[17] in normal individuals. The pressure reduction from a single drop was reported to persist for not over 6 hours.[11] Atenolol is considered less lipid soluble and more water soluble

than many β-blockers so does not penetrate the cornea readily. While no effects on corneal sensitivity were reported following short term use of atenolol eyedrops,[10] dry eyes and conjunctivitis[18] might follow longer use.

Befunolol

Befunolol, a non-selective β-blocker, was developed in Japan and is available in Germany and Japan as 0.20% (or 0.25%) and 0.5% solutions for instillation into the eye. Befunolol eyedrops have been reported to produce modest reductions in systolic blood pressure (≈10 mmHg) when used over several weeks.[19] They decreased the ability of the heart to respond to exercise[20] but had only a small effect on pulmonary function. Befunolol decreases aqueous production. Befunolol is reported to be particularly effective in eyes with various forms of secondary glaucoma.[21-23] Single drops of befunolol 0.5% promptly reduced IOP in primary glaucomatous or ocular hypertensive eyes (initial IOP ≤ 30 mm Hg) by 5 to 15 mm Hg while the 0.25% concentration produced a slightly smaller effect.[19] Both effects were maintained with either drug on a twice-daily basis.[19] Similar effects have been reported by others.[24,25] In a detailed study on patients with secondary glaucoma, slightly smaller, but long term effects, were produced[22] — again, with only slight effects on blood pressure. Its recommended clinical use would be twice-daily with the availability of two concentrations to allow titration. Befunolol is said to be comparable to pilocarpine for controlling the IOP.[21] The pressure lowering effect of befunolol can be enhanced by concurrent use of pilocarpine or acetazolamide.[23] Few unwanted systemic effects occur.[26] Overall, befunolol has been found to have only small effects on blood pressure or heart rate[19,22,24] has not so far been found to affect corneal sensitivity, outflow resistance, pupil size, nor refraction.[19,24] Other potential unwanted effects include headache, general ocular irritation and blepharitis.[26]

Betaxolol

Betaxolol is now widely available as an ophthalmic product (Canada, USA, Europe) as a 0.5% solution. This cardioselective β_1 blocker, when instilled into the eye can slow the heart (about 2 beats/min[27]) and lower the blood pressure (by 7 mmHg).[27] Betaxolol eyedrops lowered the IOP 3.8 to 11 mmHg[1,27-29] or 17% to 27%.[30-34] By itself betaxolol was not as effective as timolol in lowering the IOP when the pressure was 26 mmHg or more.[34] However other studies found equal pressure reduction by these two beta blockers.[27,35] Betaxolol is usually instilled twice a day, one drop each eye. Decisions as to the use of betaxolol are based on a variety of factors. Like the other ophthalmic β-blockers, betaxolol stings a little when first instilled.[27,29,36,37] Such irritation has been found in 22% to 70% of patients in controlled trials but betaxolol is said to produce fewer side effects than timolol.[38] When betaxolol 0.5% eyedrops are instilled bid over many weeks, the maximum reduction of IOP can be expected within 1 week and the IOP remains relatively constant thereafter[27,33,35] in at least 50% of patients placed on betaxolol as their sole glaucoma medication. Betaxolol penetrates the cornea more readily than timolol does,[27,39] and like timolol decreases aqueous production,[1,28] but is not necessarily as effective as timolol for treating open-angle glaucoma.[34] Betaxolol is racemic mixture whereas timolol consists of only the l-isomer.[37] If the dose is large enough betaxolol shows some ability to block β_2 sites and therefore causes unwanted pulmonary effects in a few patients.[40] Betaxolol has been reported to have less effect on lung function[38] and exercise-induced tachycardia than timolol does.[41] In one year there were 56 reports of adverse reactions apparently associated with use of topical betaxolol on the eye. Of the nine patients with severe reactions, seven were hospitalized, six for asthma, and one each for asthma with cardia arrhythmia, respiratory distress, and bradycardia with syncope.[42] Cardiac beta blockade has been reported.[39] Insomnia and depression have occurred. Betaxolol can also decrease corneal sensitivity and cause photophobia, increased tearing and conjunctival hyperemia[42]: the so-far reported incidence of these problems appears to be small however.

Bupranolol

Bupranolol blocks β_1 and β_2 sites and has significant membrane stabilizing activity. It is administered orally for control of cardiac arrhythmia and is not available in North America as an ophthalmic product. However this potent β_1, β_2 blocker is available in Germany (and Japan?) for ophthalmic use in concentrations of 0.05%, 0.1%, 0.25% and 0.5%.[43] Single drops of bupranolol 0.05% (in castor oil) reduced IOP by ≈5 mmHg in open angle glaucoma patients.[44] Topical bupranolol 0.5% drops (in castor oil) produced substantial (often > 10 mmHg) hypotensive effect.[43,45-47] Topical bupranolol 1% (vehicle not stated) provided a similar effect in a glaucomatous eye[46] and simultaneously lowered the IOP in the untreated eye by 70% as much as in the treated eye.[46] Bupranolol decreases aqueous production[46,47] without significant effect on aqueous outflow.[45-47] The higher concentrations of bupranolol (0.5% and 1%) have been found to be comparable to pilocarpine 2% or 4% eyedrops in ocular hypertensive patients[48] but without the unwanted ciliary spasm, miosis and tearing associated with topical pilocarpine. Bupranolol 0.5% has been tested over a period of several weeks and found to maintain its clinical efficacy.[49] Bupranolol drops can however produce significant ocular irritation and blepharitis.[49] In some patients a modest corneal anesthesia has been reported.[43,45,47] Only slight effects on tear film production have been reported.[45].

Carteolol

Carteolol is available as an ophthalmic drug in Japan, England, Italy, France and Germany at 1% or 2% concentrations. It blocks both β_1 and β_2 sites and has some membrane stabilizing action and some intrinsic sympathomimetic activity.[50] It lowers the IOP by reducing aqueous secretion.[51] In a concentration of 1% or 2% instilled three times a day, carteolol lowered the IOP by about 9 mmHg or by 34%.[50] Carteolol was reported to maintain the IOP of 84% of primary open-angle glaucoma patients below a pressure of 24 mmHg.[52] Carteolol 2% eyedrops slowed the heart an average of 6 bpm.[53] Carteolol can however cause superficial keratitis[53] to an extent that the product is marketed with the warning that it should not be used in dry eye patients as it can aggravate or precipitate this condition.

Labetalol

Although generally classified as a non-selective β_2-blocker labetalol is consi-

dered by some to also block β_1 receptors to some extent.[46] Labetalol has some membrane stabilizing activity. It is not currently available as an ophthalmic product but is taken orally to treat acute high blood pressure crises. Labetalol eyedrops (1%, 1 drop) were found to produce small and very variable reductions in IOP (3 to 6 mmHg) within 4 hours while a similar pressure reduction was observed in the contralateral eye.[54] As with most of the β-blockers, small reductions in systolic blood pressure and pulse rate have also been reported.[54] Side effects reported[18] were dry eyes and conjunctivitis.

Levobunolol

Levobunolol is a non-selective β-blocker. Orally administered levobunolol has been used to treat systemic hypertension, cardiac arrhythmia and angina. Levobunolol 0.5% is now widely available for ophthalmic use. When instilled, one drop each eye (twice a day) levobunolol 0.5% eyedrops have been found to lower IOP 2.3 to 9.6 mmHg,[55-62] or by 9% to 36%,[58,63,64] especially in patients with IOPs 30 mmHg. Some contralateral effect on IOP is also observed. The plasma level of levobunolol one hour after topical instillation of 1% was 0.3 to 0.6 ng/ml.[63] Levobunolol has a rapid onset of action and the IOP has not been found to drift higher after months of treatment. Levobunolol, instilled twice a day, is considered equivalent to timolol for treating glaucoma,[55,57,58] and like timolol decreases aqueous production.[64] Levobunolol controlled glaucoma successfully in over 70% of one group of patients.[61] When compared to other ophthalmic β-blockers levobunolol has a long duration of action[57] so there was hope that once a day administration would prove to be sufficient for controlling glaucoma in some patients[61] but other investigators consider this unlikely.[65] Levobunolol can be administered concurrently with other anti-glaucoma medications. Levobunolol slowed the heart[55,57-59] 4 to 10 beats/min[55,57,59,60] and lowered blood pressure 3 to 6 mmHg (systolic and diastolic).[33,56,60,66] Others have reported a decrease in systolic pressure of 12 to 14 mmHg and in diastolic pressure of 4 to 6 mmHg.[57] Topically instilled levobunolol can also decrease pulmonary function by as much as 25%[20] and thus, as with timolol, contraindications include: bronchial asthma, chronic pulmonary disorders, heart block, sinus bradycardia and cardia valvular disorders. Levobunolol can irritate the eyes and stinging was reported in 22% to 56% of drug-treated patients in controlled trials with 0.5% and 1%[56,66]; and has caused blepharitis,[57] or blepharoconjunctivitis in a small number of patients receiving it. Side effects occur in 1% to 4% of users of levobunolol eyedrops and include headache and insomnia. Unwanted effects are apparently more frequent in patients receiving timolol.[55]

Metipranolol

Metipranolol, a non-selective β-blocker,[20] is available in the United Kingdom, Germany and France for ophthalmic use in three concentrations: 0.1%, 0.3% and 0.6%. The 0.1% concentration is also available in Germany in combination with pilocarpine 2%. The drug was developed in Germany. FDA approval for metipranolol is currently being sought in the USA especially as the efficacy of the 0.6% concentration has been reported to be same as levobunolol 0.5%.[66] Metipranolol 0.5% or 0.3% produces a modest (5 to 7 mmHg, 15% to 30%) but sustained reduction in IOP of eyes with open-angle glaucoma.[67,68] The efficacy of metipranolol 0.25% was reported to equal that of timolol 0.25% drops in open-angle glaucoma.[69] The 0.3% or 0.6% concentration slowed the heart $\leq$ 4 beats/min,[66,68] and lowered diastolic pressure by 3 mmHg, while reducing IOP by about 3 mmHg[66] or about 5 mmHg.[66] Topical metipranolol significantly suppressed the ability of the heart to respond to exercise.[20] Topical metipranolol, like levobunolol, was found to reduce pulmonary function by 24%.[20] Because metipranolol is available in three concentrations it may permit closer control of a patient's IOP without the administration of enough drug to cause unwanted effects.[1] Metipranolol 0.25% in clinical use has not been reported to produce any more corneal anesthesia than timolol 0.25%.[70] In two studies, glaucoma or ocular hypertensive patients preferred levobunolol 0.5% to metipranolol 0.6% eyedrops when they were asked to compare how much stinging the drops produced on instillation.[66,71] In the short term, metipranolol 0.6% has been reported to produce about the same effect on tear film stability (reducing TBUT) as that produced by timolol 0.5%.[72]

Metoprolol

Metoprolol, another cardioselective β_1 blocker, is widely used to treat high blood pressure and is not currently available as an ophthalmic drug. Metoprolol has no intrinsic sympathetic activity and almost no membrane stabilizing activity.[9] Metoprolol 1% eye drops were found to reduce IOP by 4 to 6 mmHg[73] while the 3% concentration lowered IOP about 6 mmHg or about 26%.[74] It kept the pressure down for 2 to 4 hours.[74,75] Metoprolol 0.5% eyedrops have small to modest effects on heart rate with reductions of 4 bpm[76] on a bid regimen but 12 bpm on a qid regimen.[74] Metoprolol lowered systolic blood pressure by 5 mmHg[58] but produced no change in diastolic pressure. Common side effects included a burning, itching sensation in the eye[73] which resulted in increased tear production.[74] Tear breakup time has also been reported to be significantly reduced.[72]

Nadolol

Nadolol blocks β_1 sites and to some degree β_2 sites as well. By blocking β_1 sites nadolol is useful to treat cardiac arrhythmias and associated high blood pressure. It is not available as an ophthalmic product. In a recent study, nadolol 2% eyedrops instilled in the eye produced only a small and brief reduction of the IOP — an effect attributed to their low lipid solubility and thus poor corneal penetration.[77] However, earlier studies using single drops of nadolol 2% reported an average ocular hypotensive effect of at least 10 mmHg within 2 hrs and that reduction lasted for at least 4 hrs.[54,78] This effect was confirmed in a subsequent study which evaluated these eyedrops over a period of 4 weeks[79], using a twice-a-day regimen. When prepared in the form of the prodrug acetyl nadolol 0.5%, it was found to penetrate the cornea more readily and lowered the IOP about 7 mmHg.[77] Like the other β-blockers nadolol eyedrops can also slow the heart and lower blood pressure[79] but without effects on the pupil. Serious side effects of dry eyes and periorbital dermatitis have been reported however.[77]

Oxprenolol

Also called oxyprenolol this non-selective β-blocker has membrane stabilizing and

intrinsic sympathomimetic activity.[7] Oxprenolol is used to treat high blood pressure but is not currently available as an ophthalmic product. One study reported that instillation of oxprenolol drops 0.5% lowered[80] IOP about 3 mmHg (i.e. about 15%). Another trial showed single drops of 0.5% or 1% reduced IOP by an average of 6 mmHg within 2 to 3 hours. The ocular hypotensive effect was maintained for at least 3 months when oxprenolol was instilled three times a day. Oxprenolol 1.0% (1 drop) when instilled in the eye produced a slight fall in diastolic pressure and in pulse rate (by 3 to 4 beats/ min)[80] and kept the IOP down for 2 to 3 hours but tolerance tended to develop. Potential unwanted effects however include dry eyes, conjunctivitis and even corneal ulceration.[14]

Pindolol

Pindolol is a non-selective β-blocker with some intrinsic sympathomimetic activity,[7] and very little membrane stabilizing activity. It is used to manage high blood pressure. In West Germany (and France?, Italy?) pindolol is available for ophthalmic use in the 0.5% and 1% concentrations. Trials with pindolol eyedrops have produced variable results. When pindolol 0.5% or 1% was instilled in the eye it was reported to lower IOP by 3 mmHg[81] while in another trial the 0.25% concentration was found to lower IOP by 4 to 6 mmHg or about 18%.[82] Pindolol 1% eyedrops (single instillation) by 10 mmHg.[83] Similarly large reductions in IOP were reported in open angle glaucoma patients in a trial using pindolol 0.5% three times a day[84] with no change in corneal sensitivity, pupil or visual acuity in most patients. Pindolol 0.5% eyedrops, produced only small reductions in IOP (3 mmHg) and had insignificant effects on heart rate.[85] However a proportion (reported as 13% in one study[86]) of patients suffered from dry eyes, conjunctivitis, or lid reactions. These necessitated marketing the drug with a firm warning of these effects when used for a long term. This last study reported that pindolol 1% tid was effective in eyes with initial pressures of > 40 mmHg. A recent study[87] reported that pindolol 1% twice daily produced a sustained reduction of IOP of about 6 mmHg — giving it the same clinical efficacy as the commoner β-blockers such as timolol.

Practolol

Practolol blocks β_1 sites and has some ability to block β_2 sites. This drug has intrinsic sympathomimetic activity.[9] Practolol has been used to treat high blood pressure but is not now available as an ophthalmic product. Single drops of practolol 10% were found to lower IOP by 4 or 5 mmHg.[88] Practolol eyedrops however caused an intense immunological reaction (the oculomucocutaneous syndrome) with dry eyes, keratoconjunctivitis, corneal ulcers and in a few cases blindness.[89] Practolol was subsequently shown to be excessively toxic to the corneal epithelium and to the lacrimal glands. The reason for these effects remains unknown, although an autoimmune response to practolol has been suggested.[89]

Propranolol

Propranolol was the first oral β-blocker and has long been used to treat high blood pressure, angina, arrhythmia and migraine. It is a non-selective β-blocker with some membrane stabilizing activity.[9] Following initial investigations of the action of systemic propranolol (oral or i.v.) on IOP[90] propranolol 1% eyedrops were reported to produce substantial reduction in the IOP of glaucomatous eyes,[91] but the same extent of ocular hypotensive effect was not found in other studies.[92] One investigation using propranolol 0.5% reported a modest ocular hypotensive action.[93] Propranolol kept the IOP down for 4 to 6 hours but its ability to control the IOP diminished in about 2 months. It is of interest that a special study found that intranasal administration of propranolol produced a serum level equal to that achieved by intravenous administration.[94] Unwanted effects include: dry eyes, ocular discomfort and corneal anesthesia although such effects are concentration dependent.[93]

Timolol

Timolol is a non-selective β-blocker[5] which was developed to treat high blood pressure and serendipitously found to be an effective "anti-glaucoma" drug. Within the past seven years timolol has become a popular drug for treating open-angle glaucoma. It has become the highest volume sales prescription drug for ophthalmic use.[1] Timolol has very little membrane stabilizing activity and almost no intrinsic sympathomimetic activity. Timolol is available for ophthalmic use worldwide (Europe, Japan, Australia, USA and Canada). Timolol has now become the standard with which other beta blockers intended for ophthalmic use are compared. Timolol decreases aqueous production[8] by about 30%. In concentrations of 0.25% or 0.5% instilled twice a day timolol produced clinically useful reductions in IOP (2 to 10 mmHg, 18 to 30%).[27,28,35,55,57,58,61,76,77,82] The pressure was kept down for at least 6 hours and in some eyes for at least 12 hours.[9] Clinicians almost always advise twice a day instillation of timolol. Timolol appears to be absorbed more slowly and eliminated more rapidly than other β-blockers which is an unexpected finding in view of its long-lasting ocular hypotensive effect.[95] With prolonged use of timolol eyedrops, the IOP in some patients shows short term escape and (when timolol is used for 12 months or more) long term drift in the direction of higher pressure. As a result a change in medications becomes necessary. Timolol drops instilled twice a day generally produce more reduction in IOP than topical ocular epinephrine 1% twice a day. If a patient on timolol is also given epinephrine the IOP goes down 5% to 10% more. However if a patient is receiving epinephrine and is then given timolol an even greater hypotensive effect is achieved.[96] (Epinephrine itself improves aqueous outflow probably by its agonist action on β_2 adrenergic receptors.) When the goal was to keep the IOP below 21 mmHg timolol was considered satisfactory in 64% to 93.7%,[9,61] pilocarpine was considered satisfactory in 76.2%,[9] and epinephrine was satisfactory in 69.9%,[5] of patients. (Acetazolamide given orally every 6 hours will decrease aqueous production by about 40% and lower IOP 6 mmHg.[33]) Despite the proven effectiveness of timolol it should be recognized that timolol has cardiovascular effects, slows the heart 1 to 10 beats/min[27,55,57,59,60,68,76] and lowers blood pressure 2 to 10 mmHg.[27,29,55,57,60,68,76] Topically applied timolol may decrease the heart's ability to respond to exercise.[20] Timolol is contraindicated in patients with bradycardia, asthma,[28] or any kind of heart block.

Lowering the blood pressure may counteract some of the hoped-for good results of lowering the IOP since perfusion pressure in the retinal capillaries is important in preserving the health and

function of the retinal ganglion cells. The instillation of timolol (or most other β-blockers) into one eye also lowers the IOP of the contralateral, untreated eye. This is clear evidence that systemic absorption of the drug is a factor to be taken into account. Timolol has CNS effects on 10% of users and has mild pulmonary effects (reduced forced expiratory volume) on many, and as such it aggravates asthma.[38] Timolol causes fatigue, dizziness and headache in some patients and about 10% of timolol-treated patients develop depression. The external ocular effects can include dry eyes,[34] conjunctivitis and mild corneal anesthesia in a few patients[38] as well as eliciting a stinging sensation on instillation in about 20% of patients.[27,64,69]

The usual initial oral dose of timolol for treating high blood pressure is 10 mg twice a day. When one drop of timolol 0.5% is instilled in each eye the dose administered is 0.3 to 0.5 mg, twice a day. Probably at least 80% of a topically instilled eyedrop enters the vascular system more or less directly.[97] If all of the timolol 0.5% eyedrops were systemically absorbed the expected plasma concentration in a 70 kg patient would be 1.5 ng/ml. Studies have shown that the plasma concentration one hour after topical instillation of one drop of timolol 0.5% each eye was 1.3 ng/ml[97] in adults. Other studies have shown more beta blockade by timolol than by betaxolol.[98] A plasma concentration of 0.21 to 0.60 ng/ml levobunolol was reported in a similar study using levobunolol 0.5% eyedrops.[62]

Hopefully the above review will serve both as a summary and also a basis for comparison of the beta-blockers. From this comparison, it is apparent why only some of the drugs have been approved for ophthalmic use: the others show either limited efficacy (compared to timolol) or precipitate unwanted side effects at too high a frequency to be tolerated in regular use.

It can be anticipated that the proliferation of β-blockers for treating glaucoma will result in lowered costs to the patient. Possibly one of these drugs (or others being developed) will prove to be more selective, more effective, safer and even longer lasting so that once a day instillation will be sufficient as is advocated for a special pilocarpine gel (Pilopine HS) which contains pilocarpine 4%. As yet none of the β-blockers achieves all of these goals.

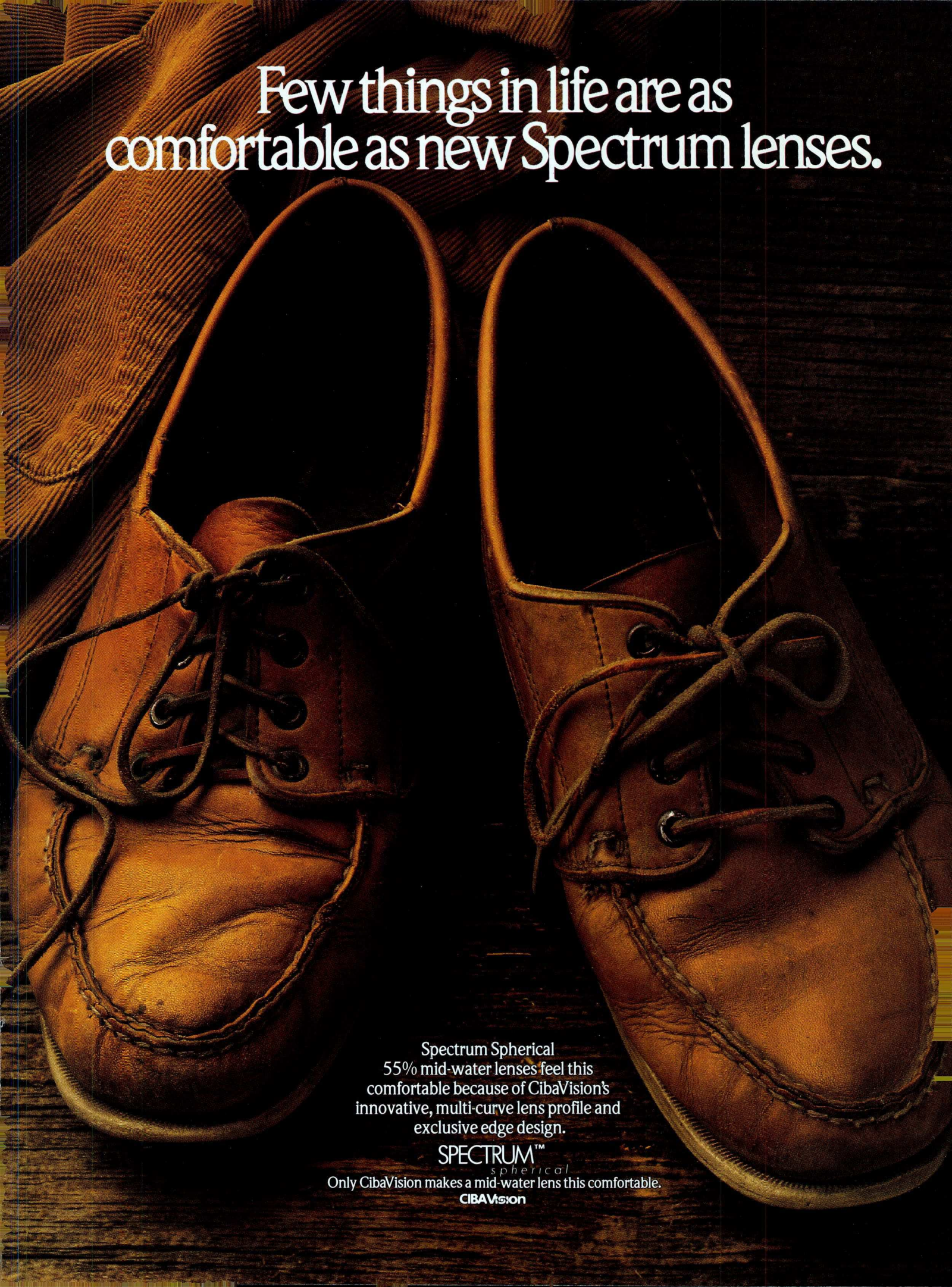
At this time, several other β-blockers are being tested for possible use as topical ocular hypotensive drugs. Such drugs include arotinol, bunolol, falintolol and soquinolol. As is evident from the bibliography in this article, β-blockers are studied and developed often on a regional basis. It is unknown at this time if any of the currently marketed or investigational β-blockers will achieve the worldwide acceptance that timolol maleate ophthalmic solution has in less than 10 years of clinical use. That such a goal is present in the minds of some is however evident from the enormous effort currently being expended in trials of ophthalmic β-blockers. An extensive listing of these clinical trials is provided in a recently published article.[99]

References

1. Doughty M.J. The timolol alternatives in open-angle glaucoma management. *Mod Probl Pharmacol* 1987; 1: 4–7.
2. AOA news — The 20th State.
3. Tierney D. W. Betaxolol and levobunolol: new beta-blocking antiglaucoma agents. *J Am Optom Assoc* 1987; 58: 722–7.
4. Bartlett J. D. Why optometrists should treat glaucoma. *J Am Optom Assoc* 1987; 58: 694–5.
5. Weiner N. Drugs that inhibit adrenergic nerves and block adrenergic receptors. In: Gilman A. G., Goodman L. S., Rau T. W., Murad F., eds. The Pharmacological Basis of Therapeutics, 7th ed. New York: MacMillan, 1985: 181–214.
6. Weiner N. Norepinephrine, epinephrine and the sympathetic amines. In: Gilman A. G., Goodman L. S., Rau T. W., Murad F., eds. The Pharmacological Basis of Therapeutics, 7th ed. New York: MacMillan, 1985; 145–80.
7. Mishima A. Ocular side effects of beta-adrenergic agents. *Surv Ophthalmol* 1982; 27: 187–208.
8. O'Donnell S. R. Review: the actions and side effects of β-adrenoceptor blocking drugs. *Aust J Optom* 1984; 67: 204–11.
9. Zimmerman T. J., Boger W. P. The beta-adrenergic blocking drugs and the treatment of glaucoma. *Surv Ophthalmol* 1979; 23: 347–62.
10. Wettrell K., Pandolfi M. Effect of topical atenolol on intraocular pressure. *Br J Ophthalmol* 1977; 61: 334–8.
11. Ros R. E., Dake C. L., Offerhaus L., Greve E. L. Atenolol 4% eye drops and glaucoma. A double-blind short-term clinical trial of a new $beta_1$ — adrenergic blocking agent. *Graefes Arch Klin Ophthalmol* 1977; 205: 61–70.
12. Wettrell K. Beta-adrenoceptor antagonism and intraocular pressure. *Acta Ophthalmol* 1977; suppl. 134; 4–54.
13. Phillips C. I., Gore S. M., Macdonald M. J., Cullen P. M. Atenolol eye drops in glaucoma: a double-masked controlled study. *Br J Ophthalmol* 1977; 61: 349–53.
14. Collignon-Brach J., Weekers R. L'atenolol, ses effects tensionnels dans le glaucome à angle ouvert. *J Fr Ophtalmol* 1978; 1: 205–10.
15. Phillips C. I., Gore S. M., Gunn P. M. Atenolol versus adrenaline eyedrops and an evaluation of these two combined. *Br J Ophthalmol* 1978; 62: 296–301.
16. Brenkman R.F. Long-term hypotensive effect of atenolol 4% eyedrops. *Br J Ophthalmol* 1978; 62: 287–91.
17. Hill S.E.W., Lewis K., Stewart-Jones J.H., Wadsworth J., Torner P. Effect of local atenolol on intraocular pressure in normal subjects using a non-invasive method. *Pharmatherapeutica* 1979; 2: 136–9.
18. Banes S.B. Beta blockers — side effects. *Optician* 1984; 187: 20–22.
19. Merte H.J., Stryz R. Erste Erfahrungen mit dem Beta-Blocker Befunolol bei Glaukomen mit weitem kammerwinkel in Europa. *Klin Monatsbl Augenheilkd* 1984; 184: 55–8.
20. Cervantes R., Hernandez Y., Hernandez H., Frati A. Pulmonary and heart rate changes associated with nonselective beta-blocker therapy. *J Toxical-Cut Ocular Toxicol* 1986; 5: 185–93.
21. Makiura M., Uyama M., Sasamoto H., Mantni M., Hattori M. Ocular hypotensive effect of an ophthalmic solution (befunolol); compared with pilocarpine. (In Japanese) *Acta Soc Ophthalmol Jpn* 1982; 86: 565–72.
22. Miki H., Shimizu Y., Nakatani H., Kinoshita A., Kosaki H. Effect of a beta-blocking agent on secondary glaucoma. (In Japanese) *Folia Ophthalmol Jpn* 1983; 34: 269–78.
23. Miki H. Effect of befunolol ophthalmic solution, an alpha, beta-adrenergic blocking agent, on secondary glaucoma. (In Japanese) *Acta Soc Ophthalmol Jpn* 1983; 87: 1–13.
24. Tane S., Komatsu A., Kubota S. Effect of befunolol ophthalmic solution (a new beta-blocking agent developed in Japan) on the intra-ocular pressure. (In Japanese) *Folia Ophthalmol Jpn* 1979; 30: 1238–40.
25. Takase M., Araie M., Matsuda T. A single dose study of topical befunolol on the intraocular pressure in man. (In Japanese) *Acta Soc Ophthalmol Jpn* 1982; 86: 87–8.
26. Shimizu Y. Effects of befunolol ophthalmic solution, a beta-adrenergic blocking agent, on ocular hypertension and primary glaucoma. (In Japanese) *Acta Soc Ophthalmol Jpn* 1982; 86: 2123–34.
27. Berry D.P., van Buskirk E.M., Shields M.B. Betaxolol and timolol. A comparison of efficacy and side effects. *Arch Ophthalmol* 1984; 102: 42–5.

28. Allen R.C., Epstein D.L. Additive effect of betaxolol and epinephrine in primary open-angle glaucoma. *Arch Ophthalmol* 1986; 104: 1178–84.
29. Feghali J.G., Kaufman P.L. Decreased intraocular pressure in the hypertensive human eye with betaxolol, a β_1 adrenergic antagonist. *Am J Ophthalmol* 1985; 100: 777–82.
30. Radius R.L. The use of betaxolol in the reduction of elevated intraocular pressure. *Arch Ophthalmol* 1983; 101: 898–900.
31. Caldwell D.R., Salisbury C.R., Guzek J.P. Effects of topical betaxolol in ocular hypertensive patients. *Arch Ophthalmol* 1984; 102: 539–40.
32. Van Buskirk E.M., Weinrib R.N., Berry D.P., Lustgarten J.S., Podos S.M., Drake M.M. Betaxolol in patients with glaucoma and asthma. *Am J Ophthalmol* 1986; 101: 531–4.
33. Smith J.P., Weeks R.H., Newland E.F., Ward R.L. Betaxolol and acetazolamide. Combined ocular hypotensive effect. *Arch Ophthalmol* 1984; 102: 1794–5.
34. Allen R.C., Hertzmark E., Walker A.M., Epstein D.L. A double-masked comparison of betaxolol vs timolol in treatment of open-angle glaucoma. *Am J Ophthalmol* 1986; 101: 535–41.
35. Levy N.S., Boone L., Ellis E. A controlled comparison of betaxolol and timolol with long-term evaluation of safety and efficacy. *Glaucoma* 1985; 7: 54–62.
36. Stewart R.H. Kimbrough R.L., Ward R.L. Betaxolol vs timolol: a six-month double-blind comparison. *Arch Ophthalmol* 1986: 104: 46–8.
37. Allen R.C., Hertzmark E., Walker A.M., Epstein D.L. A double-masked comparison of betaxolol vs timolol in the treatment of open-angle glaucoma. *Am J Ophthalmol* 1986; 101: 535–41.
38. Schoene R.B., Abuan T., Ward R.L., Beasley C.H. Effects of topical betaxolol, timolol, and placebo on pulmonary function in asthmatic bronchitis. *Am J Ophthalmol* 1984; 97: 86–92.
39. Ball S. Congestive heart failure from betaxolol. *Arch Ophthalmol* 1987; 105: 320.
40. Harris L.S., Greenstein S.H., Bloom A.F. Respiratory difficulties with betaxolol. (Letter) *Am J Ophthalmol* 1986; 102: 274.
41. Atkins J.M., Pugh B.R., Timewell R.M. Cardiovascular effects of topical beta-blockers during exercise. *Am J Ophthalmol* 1985; 99: 173–5.
42. Nelson W.L., Kuritsky J.N. Early post-marketing surveillance of betaxolol hydrochloride, September 1985 – September 1986. *Am J Ophthalmol* 1987; 103–592.
43. Stiegler G. Bupranolol-Augentropfen (Ophtorenin (R)) in der Glaukom-Dauertherapie. *Klin Monatsbl Augenheilkd* 1979; 174: 267–75.
44. Leydhecker W., Krieglstein G.K. The intraocular pressure responses flow-dose bupranolol (Ophtorenin) and methazolamide (Neptazine) in glaucomatous eyes. A controlled clinical study. *Albrecht von Graefes Arch Klin Exp Ophthalmol* 1979; 210: 135–40.
45. Krieglstein G.K., Sold-Darseff J., Leydhecker W. The intraocular pressure response of glaucomatous eyes to topically applied bupranolol. *Albrecht von Graefes Arch klin Exp Ophthalmol* 1977; 202: 81–6.
46. Sakimoto G., Une H., Ohba N. Effects of topically applied bupranolol on the intraocular pressure. Effects on the untreated eye. *Ophthalmologica* 1979; 179: 214–9.
47. Ciarnella Cantani A., D'Antino G., Genovesse S. Indagine tonografica so un nuovo farmaco ipotensivo endoculare: il bupranolo. *Clin Oculista e Patologica Oculare* 1983; 4: 43–8.
48. Totsuka H., Matsuo T., Araie M., Takase M. Studies of bupranolol therapy for glaucoma. 2. Dose response and comparing to pilocarpine in intraocular pressure. (In Japanese). *Acta Soc Ophthalmol Jpn* 1979; 83: 2166–2175.
49. Nakatani H., Sumi K-Y., Maeda K., Nakauchi M., Okabe S. Long term clinical trials in the treatment of glaucoma with topical 0.5% bupranolol, β-blockade. (In Japanese) *Folia Ophthalmol Jpn* 1979; 30: 1430–5.
50. Gorgone G., Spina F., Amantia L. Carteolol: Preliminary study on the ocular pressure-reducing action. *Ophthalmologica* 1983; 187: 171–3.
51. Araie M., Takase M. Effects of S-596 and carteolol, new beta-adrenergic blockers and flurbiprofen on the human eye: a fluorophotometric study. *Albrecht von Graefes Arch Klin Exp Ophthalmol* 1985; 222: 259–62.
52. Negishsi C., Uesugi Y., Kanai A., Nakajima A., Kitazawa Y. Studies on the treatment of glaucomatous eyes with carteolol ophthalmic solution. (In Japanese) *Acta Soc Ophthalmol Jpn* 1981; 85: 337–41.
53. Ishikawa T., Orisaka S., Hiwatari S., Taketani P., Sugimachi Y. Pilocarpine, carbachol and carteolol on open-angle glaucoma and ocular hypertension. (In Japanese) *Acta Soc Ophthalmol Jpn* 1981; 85: 837–42.
54. Krieglstein G.K., Kontic D. Nadolol and labetalol: comparative efficacy of two beta blocking agents in glaucoma. *Albrecht von Graefes Arch Klin Ophthalmol* 1981; 216: 313–7.
55. Ober M., Scharrer A., David R., Biedner B-Z., Novak G.D., Lue J.C., Robins D.S., Duzman E. Long-term ocular hypotensive effect of levobunolol: results of a one-year study. *Br J Ophthalmol* 1985; 69: 593–9.
56. Bensinger R.E., Keates E.U., Gofman J.D., Novak G.D., Duzman E. Levobunolol a three month efficacy study in the treatment of glaucoma and ocular hypertension. *Arch Ophthalmol* 1985; 103: 375–8.
57. Geyer O., Lazar M., Novak G.D., Lue J.C., Duzman E. Levobunolol compared with timolol for the control of elevated intraocular pressure. *Ann Ophthalmol* 1986; 18: 289–92.
58. The Levobunolol Study Group (Appended). Levobunolol -a beta adrenoreceptor antagonist effective in long-term treatment of glaucoma. *Ophthalmology* 1985; 92: 1271–6.
59. Cinotti A., Cinotti D., Grant W., Jacobs I., Galin M., Silverstone D., Shin D., Esters J., Lee J., Bouchey R., Novak G., Duzman E., Lue J. Levobunolol vs timolol for open-angle glaucoma and ocular hypertension. *Am J Ophthalmol* 1985; 99: 11–7.
60. Berson F.G., Cohen H.B., Foerster R.J., Lass J.H., Novak G.D., Duzman E. Levobunolol compared with timolol for the long-term control of elevated intraocular pressure. *Arch Ophthalmol* 1985; 103: 379–52.
61. Wandel T., Charap A.D., Lewis R.A., Partamian L., Cobb S., Lue J.C., Novak G.D., Gaster R., Smith J., Duzman E. Glaucoma treatment with once-daily levobunolol. *Am J Ophthalmol* 1986; 101: 298–304.
62. Novak G.D., Tang-Liu D.D-S., Kelley E.P., Liu S.S., Shen C.D., Duzman E. Plasma levobunolol levels following topical administration with reference to systemic side effects. *Ophthalmologica* 1987; 194: 194–200.
63. Partamian L.G., Kass M.A., Gordon M. A dose-response study of the effect of levobunolol on ocular hypertension. *Am J Ophthalmol* 1983; 95: 229–32.
64. Yablonski M.E., Novak G.D., Burke P.J., Cook D.J., Harmon G. The effect of levobunolol on aqueous humor dynamics. *Exp Eye Res* 1987; 44: 49–54.
65. Starita R.J., Fellman R.L. Glaucoma treatment with once-daily levobunolol. *Am J Ophthalmol* 1986; 102: 544–7.
66. Krieglstein G.K., Novak G.D., Voepel E., Schwarzbach G., Lange U., Schunck K.P., Lue J.C., Glavinos E.P. Levobunolol and metipranolol: comparative ocular hypotensive efficacy, safety and comfort. *Br J Ophthalmol* 1987; 71: 250–3.
67. Dienstbier E. Ruzickova E., Cepelik J. Metipranol v léčbě glaukomu. *Cesk Oftalmol* 1981; 37: 5–12.
68. Mills K.B., Wright G. A blind, randomized cross-over trial comparing metipranolol 0.3% and timolol 0.25% in open-angle glaucoma. A pilot study. *Br J Ophthalmol* 1986; 70: 39–42.
69. Kruse W. Metipranolol-ein neuer Betareseptoren blocker. *Klin Monatsbl Augenheilkd* 1983; 182: 582–4.

70. Draeger J., Schneider B., Winter R. Die localanasthetische Wirkung von Metipranolol in Vergleich zu Timolol. *Klin Monatsbl Augenheilkd* 1983; 182: 210–3.
71. Ober M., Scharrer A., Novak G.D., Lue J.C. Lokale subjecktive Vertraglichkeit von Levobunolol und Metipranolol in einer Doppelblind Vergleichsstudie bei Patienten mit erhohten intraokularen Druck. *Ophthalmologica* 1986; 192: 159–64.
72. Strempel I. Immediatwirkung topischer β-blocker auf die "Breakup time". *Ophthalmologica* 1986; 192: 11–6.
73. Ros F.E., Dake C.L. Negelkerke N.J.D., Greve E.L. Metoprolol eye drops in the treatment of glaucoma. A double-blind single dose trial of a $beta_1$ — adrenergic blocking drug. *Graefes Arch Klin Ophthalmol* 1978; 206: 247–54.
74. Krieglstein G.K. The long-term ocular and systemic effects of topically applied metoprolol tartrate in glaucoma and ocular hypertension. *Acta Ophthalmol* 1981; 59: 15–20.
75. Collignon-Brach J., Weekers R. Metoprolol et timolol. Etude comparative. *J Fr Ophtalmol* 1981; 275–8.
76. Neilson N.V., Ericksen J.S. Timolol and metoprolol in glaucoma: a comparison of the ocular hypotensive effect, local and systemic tolerance. *Acta Ophthalmol* 1981; 59: 336–46.
77. Duzman E., Rosen N., Lazam M. Diacetyl nadolol: 3-month ocular hypotensive effect in glaucomatous eyes. *Br J Ophthalmol* 1983; 67: 668–73.
78. Krieglstein G.K. Nadolol eye drops in glaucoma and ocular hypertension. A controlled clinical study of dose response and duration of action. *Albrecht von Graefes Arch Klin Expth Ophthalmol* 1981; 217: 309–14.
79. Krieglstein G.K. Mohamed J. The comparative multiple-dose intraocular pressure responses of nadolol and timolol in glaucoma and ocular hypertension. *Acta Ophthalmol* 1982; 60: 284–92.
80. Bucci M.G., Prescosolido N. Valutazione comparativa dell'efficacia della somministrazione locale di oxprenololo e di propranololo nella terapia del glaucoma. *Ann Ottalmol e Clin Oculist* 1979; 105: 269–76.
81. Thyas C., Stewart-Jones J.H., Edgar D.F., Turner P. The effect of 0.25% and 0.5% pindolol on intraocular pressure in normal human volunteers. *Curr Med Res Opin* 1981; 7: 550–2.
82. Andreásson S., Jensen K.M. Effect of pindolol on intraocular pressure in glaucoma: pilot study and a randomized comparison with timolol. *Br J Ophthalmol* 1983; 67: 228–30.
83. Ralli R., Rossi S., Rizzo P. Tito E. Rivalutazione del pindololo per locale come ipotonizzante oculare. *Ann Ottalmol Clin Ocul* 1983; 109: 595–601.
84. Szilvassy I., Takats I. Helyileg alkalmozott pindolol (Visken, LB 46) szemnyomascsokkento hatasa glaukomanal. *Szemeszet Ophthalmol Hung* 1978; 115: 204–8.
85. Smith R.J.H., Blamires T., Nagasubramianss, Watkins R., Poinoosawmy D. Addition of pindolol to routine medical therapy: a clinical trial. *Br J Ophthalmol* 1982; 66: 102–8.
86. Merte H-J., Stryz J.R., Mertz M. Pindolol-Augentropfen (Glauko-Visken) — Halbjahresergebrisse einer Glaukom therapie. *Klin Monatsbl Augenheilkd* 1984; 184: 227–32.
87. Stryz J.R., Merte H-J. Ergebrisse einjahrige lokaler Pindolol-Anwendung bei Weitwinkel-Glaukomen. *Klin Monatsbl Augenheilkd* 1985; 186: 43–5.
88. Vale J., Phillips C.I. Practolol (Eraldin) eye drops as an ocular hypotensive agent. *Br J Ophthalmol* 1973; 57; 210–5.
89. Rahi A.H.S., Charman C.M., Garner A., Wright P. Pathology of practolol-induced ocular toxicity. *Br J Ophthalmol* 1976; 60: 312–23.
90. Phillips C.I. Howitt G., Rowlands S.J. Propranolol as an ocular hypotensive agent. *Br J Ophthalmol* 1967; 51: 222–6.
91. Bucci M.G., Pecorigiraldi J., Missiroli A. Virno M. La somminstrazione locale del propranolo nella terapia del glaucoma. *Boll Oculist* 1968; 47: 51–60.
92. Vale J., Gibbs A.C.C., Phillips C.J. Topical propranolol and ocular tension in the human. *Br j Ophthalmol* 1972; 56: 770–5.
93. Merte H-J., Merkle W. Propranolol-augentropfen in der Glaukom-Dauertherapie. *Klin Monatsbl Augenheilkd* 1980; 177: 437–42.
94. Hussain A., Foster T., Hirai S., Kashihara T., Batenhorst R., Jones J. Nasal absorption of propranolol in humans. *J Pharm Sci* 1980; 69: 1240.
95. Schmitt C., Lotti V.J., LeDouarec J.C. Penetration of five beta-adrenergic antagonists into the rabbit eye after ocular instillation. *Albrecht von Graefes Arch Klin Exp Ophthalmol* 1981; 217: 167–74.
96. Cyrlin M.N., Thomas J.V., Epstein D.L. Additive effect of epinephrine to timolol therapy in primary open angle glaucoma. *Arch Ophthalmol* 1982; 100: 414–8.
97. Passo M.S., Palmer E.A., Van Buskirk E.M. Plasma timolol in glaucoma patients. *Ophthalmology* 1984; 91: 1361–3.
98. Bloom E., Richmond C., Alvarado J., Polansky J. Betaxolol vs timolol, plasma radio-receptor assay to evaluate systemic complications of beta-blocker therapy for glaucoma. *Invest Ophthalmol Vis Sci* 1985; 26(Suppl): 125.
99. Novak G.D. Ophthalmic beta-blockers since timolol. *Surv Ophthalmol* 1987; 31: 307–27.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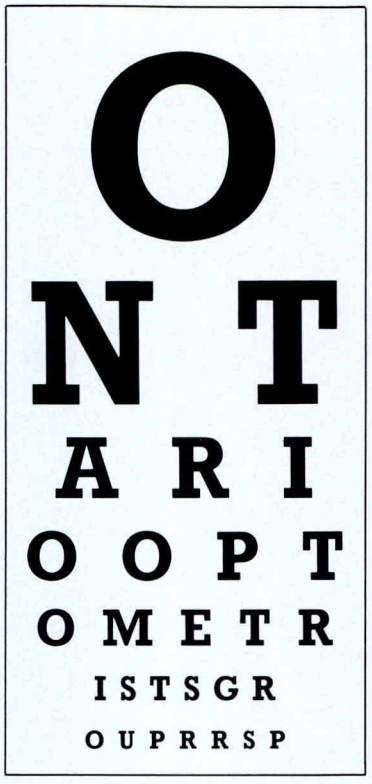

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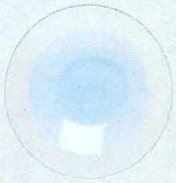

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Case Report: Congenital Drusen of the Retina and Hypertrophy of the Retinal Pigment Epithelium in the Same Eye

T.D. Williams *

The patient is a 24 year old caucasian female with no history of ocular or systemic disease and normal visual acuity in each eye. The right eye was unremarkable, both centrally and peripherally. In the left foveal region a number (roughly a dozen) of orange-colored, discrete circular spots were seen in the deep retina (Fig. 1, upper). These spots showed a tendency to glow in retro- or indirect illumination. Their diameter was on the order of 30–40 micrometers. These findings are consistent with retinal drusen. In the area superior to the fovea, a number of similar-sized spots of increased RPE pigmentation were seen, in proximity to the branches of the temporal retinal vessels.

In the inferior retina of the same eye, slightly posterior to the equator, a number of large pigmented areas were found (Fig. 1, lower). The shape (elliptical, with the long axis pointing toward the posterior pole), size, color and flatness of these pigmented areas, coupled with the absence of any corresponding scotomas are consistent with hypertrophy of the retinal pigment epithelium[1]. These pigmented lesions are benign. While perimetric testing of the left eye revealed no abnormalities, there were a few, rather variable disturbances in the Amsler grid field of the left eye: these consisted of one or two areas (each roughly one degree in diameter) of slight blurring of the lines within one degree of the fixation point. There were no scotomas.

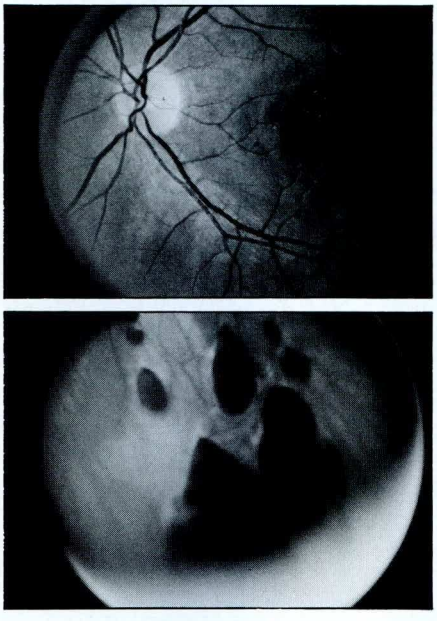

FIGURE 1

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Comment:

The role of the retinal pigment epithelium (RPE) in the genesis of localized thickenings of Bruch's membrane (drusen) has been described in detail by Farkas et al[2], Krill et al[3], Gass[4], and more recently by Ishibashi et al[5]. These authors all agree that the basis of retinal drusen (whether of the congenital or acquired variety) is some degree of abnormal RPE function, leading to deposition of hyaline-like material on the underlying Bruch's membrane.

In his longitudinal study of 200 patients with drusen and disciform macular detachment and degeneration, Gass[4] noted that approximately 33% of the patients showed large pigment epithelial cells in the equatorial area of the fundus, frequently adjacent to retinal drusen. The present case appears to show a corresponding appearance at the posterior pole.

In the last twenty years, retinal drusen have been found in association with a surprisingly wide range of ocular and systemic anomalies. These include: drusen of the optic nerve head[3], peripheral areas of enlargement of RPE cells[4], and pseudoxanthoma elasticum[3,6]. Drusen of the nerve head, in turn, have been found in association with intracranial tumors (such as craniopharyngioma and chromophobe adenoma)[7], mesodermal dysgenesis of the optic nerve head[8], and minimal brain dysfunction (clumsiness, delayed speech development, learning difficulties later in life)[9].

The findings of this case serve to remind the clinician that the retinal pigment epithelium may participate in formation not only of retinal drusen but also of sometimes dramatic (but in this case benign) peripheral pigmented areas.

References

1. Buettner H. Congenital hypertrophy of the retinal pigment epithelium. *Am J Ophthalmol* 1975; 79(2): 177–189.
2. Farkas T.G., Krill A.E., Sylvester V., Archer D.B. Familial and secondary drusen: histological and functional correlations. *Trans Am Acad Ophthalmol and Otol* 1971; 75(2): 333–43.
3. Krill A.E., Klien B.A., Archer D.B. Precursors of angioid streaks. *Am J Ophthalmol* 1973; 76(6): 875–9.
4. Gass J.D.M. Drusen and disciform macular detachment and degeneration. *Arch Ophthalmol* 1973; 90(3): 206–217.
5. Ishibashi T., Patterson R., Ohnishi Y., Inomata H., Ryan S.J. Formation of drusen in the human eye. *Am J Ophthalmol* 1986; 101(3): 342–53.
6. Erkkila H., Raitta C., Niemi K.M. Ocular findings in four siblings with pseudoxanthoma elasticum. *Acta Ophthalmol* 1983; 61: 589–99.
7. Mustonen E. Optic disc drusen and tumors of the chiasmal region. *Acta Ophthalmol* 1977; 55: 191–200.
8. Mullie M.A., Sanders M.D. Scleral canal size and optic nerve head drusen. *Am J Ophthalmol* 1985; 99: 356–9.
9. Rantala S.L., Santavuori P., Erkkila H., Riska T.B. Phoniatric and neurological findings in children with optic disc drusen. *Folia Phoniatr* 1983; 35: 316–321.

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Visual Dysfunction In Recent Onset Diabetes: A Clinical Report

J.V. Lovasik *
A.C. Kothe **

Abstract

A clinical report is presented of a 64 year old patient with recent onset diabetes. Simple in-office test procedures for the detection of subtle functional abnormalities in the visual system are presented. Diagnostic findings are expanded by results of electrophysiological testing, and complemented by an investigation of vascular structural integrity by fluorescein angiography. The importance of an examination routine consisting of an assessment of both functional and structural aspects of the visual elements typically affected by the diabetic condition is emphasized.

Introduction

The effects of diabetes on the visual system range from abnormalities in the structure and action of the extraocular muscles, abnormalities in pupil function, dark adaptation, and intraocular pressure, and anatomical alterations within the iris, crystalline lens, and retinal vasculature. Generally, changes in the morphology of ocular tissues are thought to occur only after the patient has been diabetic for many years. The alteration of ocular anatomy appears to occur independent of the degree of normalization and stabilization of blood-glucose levels. Although it is generally agreed that adequate control of blood-glucose levels will delay the onset of physical changes within ocular structures, there is insufficient clinical data to conclude that tight control of blood-glucose levels will prevent the onset of the most devastating consequence of diabetes on the eye, namely diabetic retinopathy. In fact, some preliminary clinical trials utilizing insulin pumps to accurately control blood-glucose levels have shown an acceleration of retinopathy in patients switched from traditional insulin injection procedures to the insulin pump (Begg, 1984). However, these findings are only preliminary and should not be interpreted as inevitable blindness for the patient regardless of the degree of control of the diabetic condition.

Woodruff et al. (1983) and Spafford and Lovasik (1986) have clearly demonstrated functional defects in the juvenile diabetic population prior to significant alterations in intraocular structures. The present report illustrates the utility of simple, but all too often underutilized, in-office techniques for the detection of functional abnormalities in the visual system, and their congruence with data provided by more elaborate electrophysiological testing as well as invasive procedures such as fluorescein angiography. In addition to the standard test procedures forming an oculo-visual assessment, tests of functional reserves can be administered to the diabetic patient in an effort to correlate these findings with anatomical changes observed in the visual system.

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Patient History

A 64 year old, Caucasian female was referred by a chapter of the Canadian Diabetes Association to the Electrodiagnostic Clinic at the University of Waterloo for an assessment of visual function. The patient was a diagnosed diabetic for three years and was medically managed by 32 units of insulin per day. The effectiveness of insulin in controlling blood-glucose levels was monitored by urinalysis. The patient was in generally good health and suffered only minor health problems related to arthritis. At the time of the examination the patient reported her blood-glucose levels to be normal.

The patient's ocular history indicated that she experienced somewhat impaired vision since her diagnosis of diabetes. A visual assessment some six months earlier by an optometric practitioner did not disclose any significant diabetic macular signs or age-related changes. However, vascular abnormalities typical of diabetes were found in more peripheral areas. The patient also reported fluctuating vision when blood-glucose levels were high.

Relevant visual and ocular findings at the patient's first visit were as follows:

Visual Acuity (Aided)

	OD	OS
6m	6/9	6/7.5
0.4m	0.5M	0.37M

Ocular Motility

Eye movements were unrestricted in all cardinal positions of gaze. The patient was non-strabismic.

External Findings

The ocular tissues and adnexa were considered normal for the patient's age. There were no signs of diabetic involvement in any of the tissues within the anterior segment of the eye.

Ophthalmoscopy

Direct ophthalmoscopy was employed to allow visualization of minute and subtle alterations of retinal vasculature. Indirect ophthalmoscopy lacks the optical magnification needed to see some of the finer alterations of structure found in diabetes. An overall impression of fundus changes associated with diabetes was obtained during fundus photography. Both eyes showed distinct background diabetic retinopathy. This consisted of numerous dot hemorrhages located primarily within the macular and paramacular areas. The right eye had numerous hard exudates in the macular area and a large serous exudate supero-nasal to the macula. Intraretinal microvascular anomalies (IRMA) were evident in both eyes. Both eyes showed venous beading. Significant

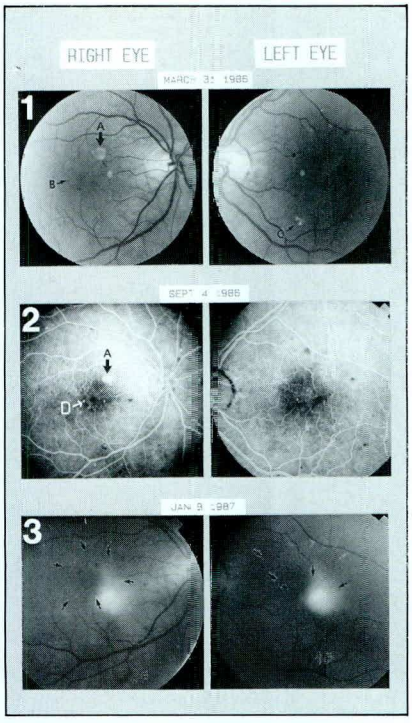

Figure 1:
Plate 1: Fundus appearance of right and left eyes at the posterior pole. Arrow A shows a large exudate at the one o'clock position referenced to the macula in the right eye. Many dot hemorrhages (for example, arrow B), as well as hard exudates (arrow C) are seen in both eyes.
Plate 2: Choroidal phase fluorescein angiograms taken about five months after the initial visit. Arrow A characterizes the lesion shown in Plate 1 as a retinal pigment epithelium detachment. Note the increased visibility and density of microaneurysms in the fluorescein angiograms compared to standard fundus photographs. Microaneurysms are seen as tiny hyperfluorescent lesions in the macular areas of both eyes (arrow D).
Plate 3: Appearance of the posterior poles two and a half months following laster photocoagulation treatment in the parmacular areas of both eyes. The small arrows point towards therapeutic focal argon lesions distributed in a roughly circular pattern in the right eye and less regularly in the left eye. Note the disappearance of the large circular lesion (Arrow A in plates 1 and 2) following treatment. Note also the occurrence of a new flame shaped hemorrhage in the right eye (arrow E) and IRMA in the left eye (arrow F). The diffuse grey spots near central maculae are artifacts of photography.

macular edema was not evident in either eye. The abnormalities seen on ophthalmoscopy are shown in Figure 1, plate 1.

The density of dot hemorrhages and exudative material in the macular area suggested a greater impairment of visual function than was measurable by the relatively small reduction in visual acuity in each eye. Several simple tests were selected to disclose further functional defects. These tests and results are summarized below.

1. Macular Photostress Test:

This test is primarily used to differentiate a maculopathy from a neuropathy as the cause for a reduction in visual acuity (Lovasik, 1983). Briefly, it involves measuring the time required to read two or more letters in the best acuity line after a macular dazzling period. The time for recovery from such a photostress period is dependent upon the structural and functional integrity of the macular area. Normal photostress recovery times are obtained for patients whose cause of

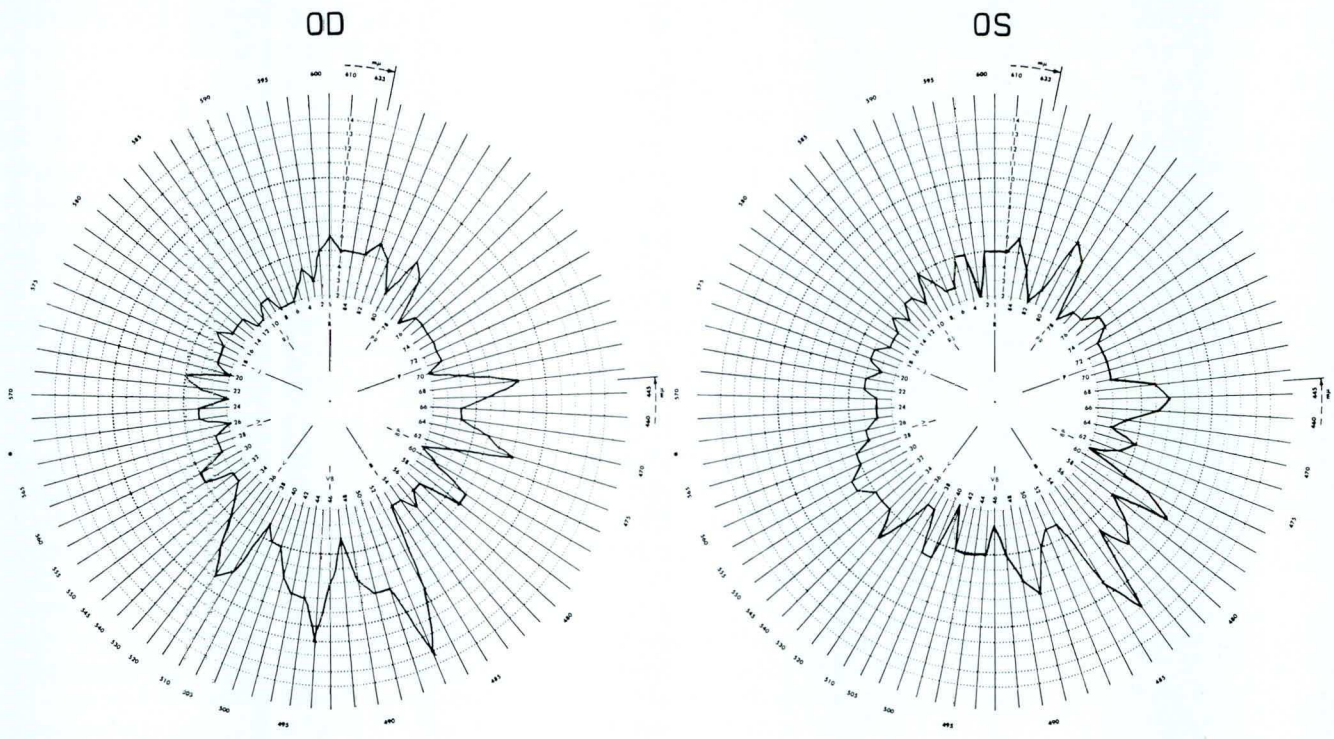

Figure 2:
FM-100 Hue error score recordings for the right and left eyes. Error scores were 214 and 218 for the right and left eye, respectively. Poor colour discrimination was inferred although no specific axis was indicated.

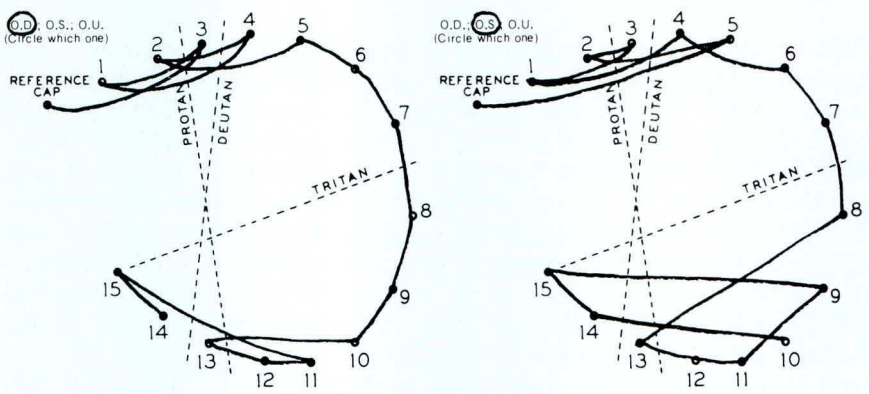

Figure 3:
Desaturated panel D-15 scoring for the right and left eyes illustrating frequent minor reversals with a tendency towards a tritanomalous defect.

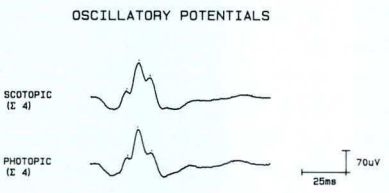

Figure 5:
The normal number of oscillatory potentials in our testing protocol were recordable for both eyes under scotopic and photopic conditions. Temporal and amplitude characteristics of these potentials were considered to be within normal limits. Recordings represent responses from the left eye. Dots identify the first, second, and third oscillatory potentials.

decreased acuity is optic nerve in origin since photochemical processes involved in the recovery of vision from bleaching are normal. Vascular or structural anomalies within the macula affecting vision also affect the photopigment regeneration process and consequently result in a prolonged photostress recovery time. For our patient the photostress times were OD 129 sec, OS 364 sec. Both values were far above the normal range of responses for the test as applied at our clinic. An upper normal value for the procedure used here is considered to be approximately 30 sec. The large difference in photostress recovery times between the two eyes highlights the fact that large functional deficits may exist between eyes with similar visual acuities and grossly similar fundus appearance.

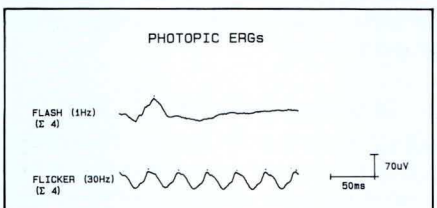

Figure 6:
Sample recordings of photopic ERGs to red flash and red flicker stimulation, indicating normal cone function. Records represent the average of 4 and 8, 200ms epochs, respectively. Identical responses were obtained from the two eyes. Peaks at which amplitudes were measured are denoted by dots (.).

2. Colour Vision Assessment:

Acquired ocular diseases frequently affect colour perception. With this in mind, both the FM-100 Hue and the desaturated panel D-15 were administered monocularly to the patient. The results of this testing are illustrated in Fig. 2 and Fig. 3 for the 100 Hue and the panel D-15, respectively. The patient made frequent colour reversals of a general nature with a tendency to show a tritanomalous colour defect, an anomaly frequently seen in various disease processes of the retina including diabetes (Adams et al, 1987).

3. Prism Competition Test:

This test is primarily utilized to identify and differentiate the laterality of a prechiasmal or post-chiasmal lesion (Mehdorn, 1980). Briefly, this test involves observing eye movements when base out and base in prisms (four prism diopters each eye), are quickly placed before the eyes as the patient views a distant fixation target. Normal responses include convergence, divergence, or alternate fixation when the total prism value before the eye exceeds fusional reserves. Positive responses include sustained dextroversion or laevoversion with prisms in place. Pre-chiasmal lesions result in the

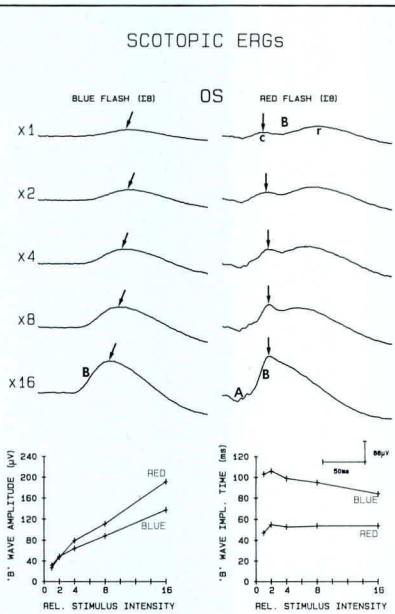

Figure 4:
Scotopic ERGs to graded relative intensities (×1 to ×16) of scotopically matched blue and red flash stimuli for the left eye. Each record represents the average of eight ERGs. Similar responses were seen for the right eye. Blue flash responses (rod isolated) demonstrated an increased amplitude and decreased implicit time of the b-wave component with increased flash intensity. The implicit times are identified by small oblique arrows. Red flash responses (rod dominated) demonstrated an increased amplitude and relatively constant implicit time for the cone component (c) of the biphasic b-wave with increased flash intensity. The implicit times for the cone component are shown by vertical inverted arrows. A rod/cone break is evidenced by the double peak within the b-wave at low flash intensities. The amplitudes of the b-wave component were measured from the trough of the a-wave to the peak of the b-wave. c = cone component, r = rod component in red flash ERG recordings.

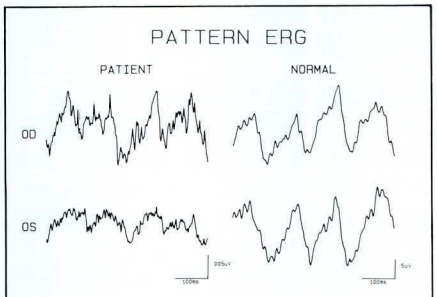

Figure 7
Pattern ERG recordings for the right and left eyes of the patient as compared with an age- and sex-matched non-diabetic subject. Each record represents the average of 100 epochs. Note the difference in the vertical scales for the patient and the normal subject. Note also the inferior response from the left eye of our patient.

superior eye determining the direction of the versional response according to whether base in or base out prisms are placed before the eyes. Post-chiasmal lesions result in sustained dextroversion or laevoversion when either direction of prisms are placed before the eyes, with the laterality of the version identifying the side with the post-chiasmal lesion. Thus, a post-chiasmal lesion on the right hand side results in a dextroversion with either base in or base out prisms. For our patient, the test results were positive for a pre-chiasmal lesion on the left hand side and were in agreement with a prolonged photostress recovery time for the left eye. Thus even though visual acuities were similar for each eye, there were demonstrable differences in neural conduction times between the two eyes.

4. Electrophysiological Tests:

In addition to these simple tests, an electrophysiological assessment of visual function was performed. Retinal function was evaluated by flash (fERG) and pattern (pERG) electroretinograms (Lovasik & Kothe, 1986). The integrity of the macular-cortical fibres were assessed by flash and pattern visually evoked responses (VERs) (Lovasik & Woodruff, 1983). The results of scotopic fERG testing for the left eye by scotopically matched blue and red flashes of increasing intensity are shown in Fig. 4. Although the implicit times of the b-wave component of the ERG were somewhat delayed and the amplitudes reduced, they were not diagnostic for diabetic retinopathy. The amplitude and implicit time characteristics of the b-waves were not out of range with those reported by Weleber (1981) for patients in the seventh decade of life. The oscillatory potentials (Speros & Price, 1981), an electrical index of the function of the inner plexiform layer of the retina, were present and not considered abnormal. Sample recordings for the left eye are shown in Fig. 5. Photopic flash and flicker ERGs indicated normal gross cone function in each eye. Sample data for the left eye are shown in Fig. 6. Similar responses were obtained for the right eye under these test conditions.

Pattern ERGs, elicited by a reversing checkerboard target, were obtained for each eye and compared to an age- and sex-matched non-diabetic subject. These results are presented in Fig. 7. The pERG morphology was relatively undisturbed but the amplitude of the signal was far below normal values in each eye, more so for the left eye than for the right. In as much as the pERG is thought by some to reflect the function of the retinal ganglion cell layer, our results pointed to a possible retinal dysfunction at the most vitread layer of the retina.

A detailed electrophysiological assessment of macular function was also performed by steady state (ssVER) and transient (tVER) pattern visual evoked responses. These were elicited by a checkerboard target reversing at 8 and 2 Hz, respectively. The results for ssVERs for the right and left eyes of the patient and an age-matched non-diabetic subject are shown in Fig. 8. The calibration scale for each record indicated a profound reduction in amplitude of our patient's ssVERs. This identified highly reduced reactivity of macular-cortical neurons tuned for spatial resolution. Given the ophthalmoscopic appearance of both maculae, the gross attenuation of the VERs was likely of retinal origin.

The amplitude of the tVER was also severely reduced and the implicit time of the P-100 component for the left eye was delayed relative to the right eye. However, both implicit times were considered to be within a normal range. Transient pattern VERs for our patient are shown in Fig. 9 and compared with an age-matched non-diabetic subject.

The ability of the macular-cortical pathways to relay information related to simple light detection was tested by the determination of the cortical critical frequency of photic driving (CFPD) (Celesia & Daly, 1977). The VER amplitude-flash frequency function is graphically illustrated in Fig. 10. Normal CFPD values exceed 40 Hz. For our patient, both eyes

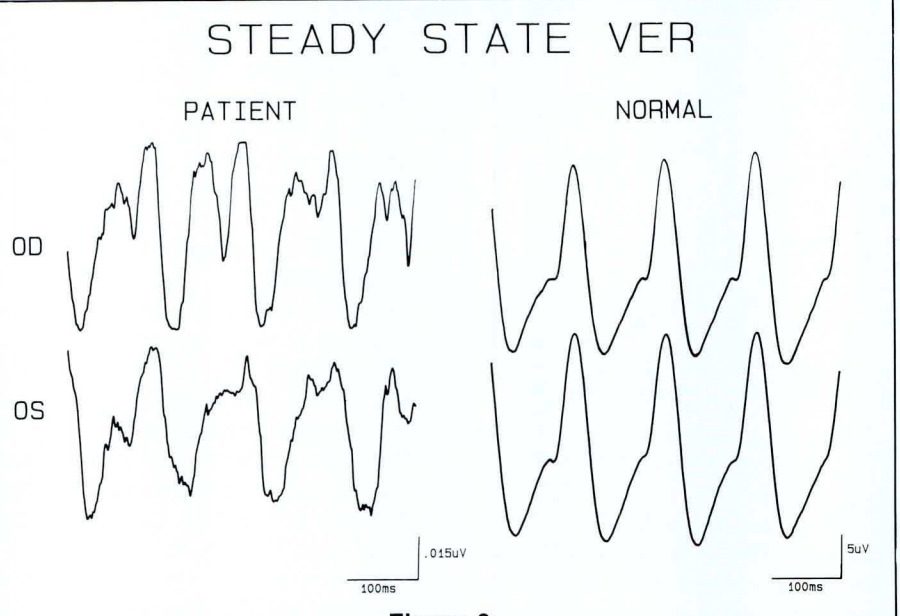

Figure 8:
Steady state VERs to a 6 degree diameter reversing (B Hz) checkerboard composed of high contrast 14 minutes of arc black and white checks. Each waveform is the average of 30 epochs. The VERs of the patient are compared to those for an age- and sex-matched non-diabetic subject. Whereas the waveforms are not significantly different, the patient's responses are many times smaller than those for the visually normal subject. Note the difference in the vertical calibration scales.

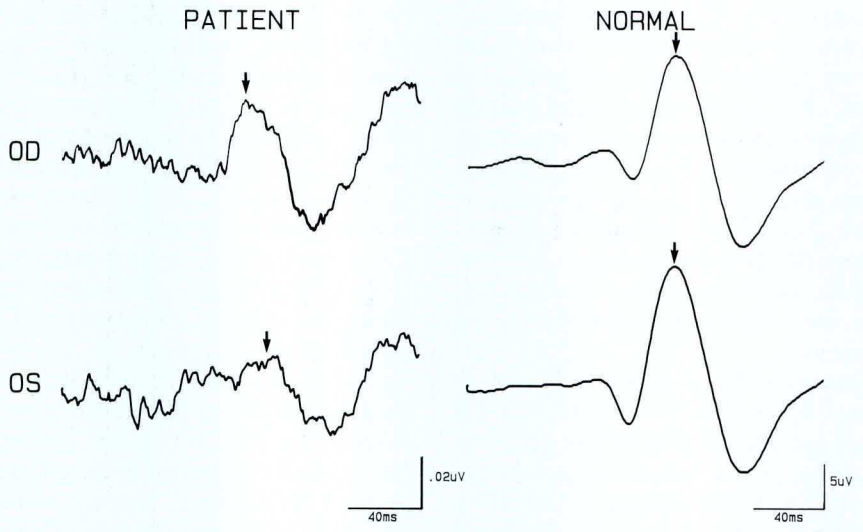

Figure 9:
Transient VERs to a 6 degree reversing (2 Hz) checkerboard composed of high contrast 14 minutes of arc black and white checks. Each waveform is the average of 30 epochs. The right and left eyes of the patient are compared with those of an age and sex-matched non-diabetic subject. As for the ssVERs, there was a very large difference in the amplitude of the evoked potential in relation to a non-diabetic subject. Note the difference in the amplitude scales for the patient and the normal subject. The implicit times for the P-100 component of the pattern VER are shown by small inverted arrows. The left eye of the patient showed a slightly delayed response when compared with the right eye. This difference was not considered diagnostically significant.

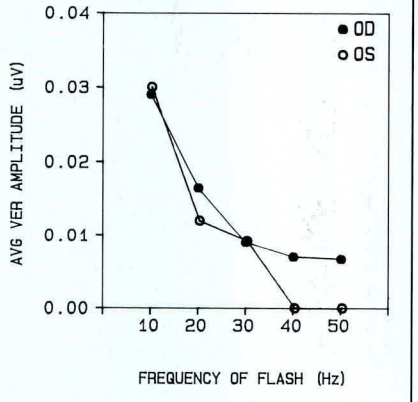

Figure 10:
Cortical CFPD amplitude-flash frequency relationship for the right and left eye of the patient. The flash VER amplitude is far below normal. A normal observer would continue to respond up to those flash frequencies establishing the psychophysically measured critical fusion frequency.

failed to conduct flash information much beyond 40 Hz. Furthermore, the response amplitude was exceptionally low, a finding consistent with all other test results signalling a significant maculopathy.

5. Ophthalmological Diagnosis/ Treatment:

An ophthalmological evaluation performed about three months after our initial assessment also concluded with a diagnosis of moderately severe non-proliferative background diabetic retinopathy. Fluorescein angiography done two months later revealed abnormal choroidal and retinal vascular filling phases for both eyes, with multiple tiny hyperfluorescent lesions located throughout the posterior pole. Both eyes exhibited mild macular edema. The right eye was generally better than the left but showed an area of retinal pigment epithelium detachment supero-nasal to the macula. The fluorescein angiograms are shown in Figure 1, plate 2. Focal argon laser therapy was carried out one and a half months later on both eyes in an attempt to minimize the progression of the diabetic retinopathy.

6. Followup Examination:

On a return visit approximately two and a half months after laser therapy, visual performance was assessed by visual acuity, VERs, colour vision and the photostress recovery test.

Aided visual acuity had improved in the right eye from 6/9 to 6/7.5 while the left eye gained two letters yielding 6/7.5 +2. The patient felt that her vision in the left eye was notably better than that for the right eye even though measured acuities were similar.

While the morphology of all pattern evoked potentials was unchanged from the initial evaluation and conformed to VERs recordable from a visually normal patient, the amplitudes had increased some ninety times. Flash evoked cortical potentials showed an even greater increase in amplitude but the associated cortical CFPD remained unchanged from that found prior to laser therapy.

Colour vision, as assessed by the desaturated panel D-15 remained unchanged from the previous visit. A tendency for a blue-yellow defect was still evident in both eyes.

The photostress recovery time for the left eye showed a dramatic improvement (first visit 364 sec, post-laser therapy 134 sec). Curiously, the right eye, that showed improved visual resolution, now showed reduced functional reserves (first visit 129 sec, post-laser therapy 215 sec). These latter observations indicated continued sub-clinical disturbances of the macula when the photochemical processes of vision were stressed by bleaching. The need for continued monitoring of this patient was strongly indicated.

Discussion

This clinical report is presented to emphasize the optometrist's role in managing the diabetic patient. Several simple in-office procedures are presented to demonstrate their use in the detection of subtle functional abnormalities in the diabetic visual system. Their proper application can lead to the required referral and management of the often hidden deficits.

Eye practitioners are reminded that appropriate colour vision testing should be routinely employed with diabetic patients. Kollner's 'rule' predicts that blue-yellow defects result from lesions of the outer retinal layers while red-green defects develop from lesions of the inner

retinal layers and optic nerve. Changes in colour vision with retinopathy have been reported in diabetics, particularly blue-yellow defects (Adams et al, 1987), which may worsen or improve as the underlying pathology progresses or undergoes remission. The FM-100 Hue test, although time-consuming, is especially effective in detecting acquired colour vision defects. However, the desaturated D-15 panel is much quicker to administer and is sensitive in detecting mild acquired colour vision defects, more so than the commonly used standard D-15. Optometrists are therefore encouraged to administer tests specific for detecting mild colour vision defects to all their diabetic patients on a routine basis.

The photostress recovery test is also a simple procedure which can be administered to all diabetics. The recovery time of this test is dependent on the rate of photopigment resynthesis and the functional integrity of the outer retinal layer and retinal pigment epithelium (Lovasik, 1983), giving additional diagnostic information in a case of suspected maculopathy.

These tests can be further complemented by the prism competition test and careful funduscopic examination and photodocumentation. In-office monitoring of blood-glucose levels can identify those patients who erroneously or falsely report good control of blood-glucose levels. In this regard it should be noted that more accurate estimates of blood-glucose levels are obtained from blood samples by simple pin prick than by measures based on urinalysis (Begg, 1984). Furthermore, since many diabetics experience their disease-related colour vision defects, it is advisable to utilize digital glucometers rather than colour matching of diagnostic sticks to colour coded reference standards in order to establish blood-glucose levels (Shute & Oshinskie, 1986).

Although more commonly utilized in group practices, hospitals and educational institutions, electrophysiological tests often provide clinically useful information concerning neural function in the diabetic patient prior to any ophthalmoscopically visible alterations in structure. The oscillatory potentials have been shown to be useful in predicting the rate of progression of diabetic retinopathy (Bresnick, 1984; Speros and Price, 1981). Others (Arden et al, 1986) maintain that the pattern ERG is even more useful clinically in the management of diabetics since it is apparently attenuated at the stage of diabetic retinopathy when referral for laser treatment becomes necessary. Pattern VERs are specific in detecting functional abnormalities in the macular-cortical pathways (Sokol, 1976; Sherman, 1979) and may be affected when retinopathy encroaches onto the macular area or when diabetic ischemic processes have affected retrobulbar components of the visual system.

In addition to drawing the reader's attention to the testing resources available for the clinical care of diabetic patients, the authors wish to dispel the notion that diabetes does not affect the visual system until the patient has been diabetic for many years. Current research indicates that functional changes in the visual system of the diabetic patient may occur in as little as five years in either juvenile or adult populations (Spafford & Lovasik, 1986). The present case is that of a patient who experienced visual abnormalities and incurred structural damage after being diagnosed diabetic for as little as three years. Structural changes were best visualized by fluorescein angiography. As is frequently the case in age-onset diabetes, compared with the rather abrupt onset of juvenile diabetes, our patient may unknowingly have been diabetic for a considerably longer period of time. However, it is also possible that diabetes acquired later on in life results in an acceleration of age-related changes in the visual system.

In summary, this report emphasizes the special role of the optometrist in the provision of eye care to the diabetic population. Vision testing should include tests routinely performed in an oculo-visual assessment, as well as those measuring functional reserves. With the exception of fluorescein angiography, the tests described here can be readily performed in the optometric office and will enhance the diagnostic capabilities so important in the management of diabetic patients.

Acknowledgements

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References

1. Adams A.J., Schefrin B., Huie K. 1987. New clinical colour threshold test for eye disease. *Amer J Optom Physiol Optics* 64(1): 29–37.
2. Arden G.B., Hamilton A.M.P., Wilson-Holt J., Yudkin J.S., Kurtz A. 1986. Pattern electroretinograms become abnormal when background diabetic retinopathy deteriorates to a preproliferative stage: possible use as a screening test. *Brit J Ophthalmol* 70: 330–335.
3. Begg I.S. 1984. Diabetic retinopathy: a review of the general medical factors in patient care. *Can J Ophthalmol* 19(4): 159–168.
4. Bresnick G.H., Korth K., Groo A., Palta M. 1984. Electroretinographic oscillatory potentials predict progression of diabetic retinopathy. Preliminary report. *Arch Ophthalmol* 102: 1307–1311.
5. Celesia G., Daly R.F. 1977. Effects of aging on visual evoked responses. *Arch Neurol* 34: 403–407.
6. Lovasik J.V. 1983. An electrophysiological investigation of the macular photostress test. *Invest Ophthalmol Vis Sci* 24: 437–441.
7. Lovasik J.V., Kothe A.C. 1986. The pattern evoked electroretinogram: Origin, characteristics and clinical usage. *Can J Optom* 48(1): 28–42.
8. Lovasik J.V., Woodruff M.E. 1983. Increasing diagnostic potential in pediatric optometry by electrophysiological methods. *Can J Optom* 45(2): 69–83.
9. Mehdorn E.T. 1980. The prism competition test. Presented at the Association for Research in Vision and Ophthalmology. Orlando, Florida.
10. Sherman J. 1979. Visual evoked potential (VEP): basic concepts and clinical applications. *J Amer Optom Assoc* 50(1): 19–30.
11. Shute D.T., Oshinskie L. 1986. Acquired colour vision defects and self monitoring of blood sugar in diabetics. *J Amer Optom Assoc* 55(11): 824–831.
12. Sokol S. 1976. Visual evoked potentials: Theory, techniques and clinical applications. *Surv Ophthalmol* 21(1): 18–44.
13. Spafford M.M., Lovasik J.V. 1986. Clinical evaluation of ocular and visual functions in insulin-dependent juvenile diabetics. *Amer J Optom Physiol Optics* 63(7): 505–519.
14. Speros P., Price J. 1981. Oscillatory potentials. History, techniques and potential use in the evaluation of disturbances of retinal circulation. *Surv Ophthalmol* 25(4): 237–252.
15. Weleber R.G. 1981. The effect of age on human cone and rod ganzfeld electroretinograms. *Invest Ophthalmol Vis Sci* 20(3): 392–399.
16. Woodruff M.E., Lovasik J.V., Spafford M.M. 1983. Ocular accommodation in juvenile diabetics: A preliminary report. *Can J Optom* 45(3): 146–149.

Diagnostic and Therapeutic Considerations In An Amblyopic Child: A Case Report

L. Sorbara *
M.M. Spafford * *

Abstract

Degraded retinal imagery and concomitant abnormal binocular interaction, as a result of significant unilateral uncorrected refractive error, can result in unilateral functional amblyopia in young humans.[1-7]

The "plastic or critical period of vision" could be considered as the physiologic epoch in which visual deprivation can impede complete development of normal vision or in which visual stimulation can "reverse" the effects of previous stimulus deprivation. This plastic period has been defined in some mammals[8,9] *but is still approximated in humans.*[8,10,11] *The prevention of normal visual acuity development with maturation is referred to as amblyopia of arrest; a condition that is non-treatable by definition.*[3] *Amblyopia of extinction represents a reduction in acuity from that previously developed and consequently acuity is recoverable.*[3] *The clinical differentiation of amblyopia of arrest from amblyopia of extinction would be greatly simplified if the critical plastic period in humans was more clearly defined.*

Optometrists must assess many factors in addition to age when evaluating the prognosis for amblyopia therapy.[11] *This case study helps illustrate the importance of early detection and subsequent correction of unequal refractive errors as well as the aniseikonic, binocular and electrophysiologic consequences of the condition and its treatment.*

History

Three year old AR and her fraternal twin were brought by their parents to Primary Care Services at the University of Waterloo, School of Optometry for their first oculo-visual assessment in June of 1984.

The 26 year old mother reported that throughout her pregnancy she suffered no illnesses, took only occasional aspirin, but smoked approximately three cigarettes a day. The instrument birth of the twins was without complications. AR weighed 5 lbs. 14 oz. at birth. Her general mental and physical development had been unremarkable. No visual problems were obvious to the parents.

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Diagnostic Findings

An oculo-visual assessment of AR's twin sister revealed normal monocular and binocular acuities, gross binocularity, unremarkable ocular health as well as insignificant and equal hyperopia in each eye.

An initial assessment of AR uncovered a gross acuity reduction in the right eye only. A significant amount of myopia was revealed in the right eye by retinoscopy and ophthalmoscopy (approximately −13 DS). The left eye was slightly hyperopic (+0.75DS).

An internal referral to Electrodiagnostic Services was arranged to determine the functional integrity of the macular-cortical pathways and the prognosis for amblyopia therapy. At that assessment the reduction in acuity and the presence of significant anisometropia were confirmed. Fixation was centred by ophthalmoscopic visualization.

Unpatterned flicker Visually Evoked Responses (VERs) were recorded with a bipolar surface electrode configuration placed along the occipital mid-sagittal plane. The active electrode rested approximately 2 cm above the inion with the reference electrode 4 cm higher. The right earlobe was grounded. VERs were recorded to white flashes (Grass stimulator X16 intensity) presented in a ganzfield at 10, 30 and 50 Hz. Thirty 200 ms epochs

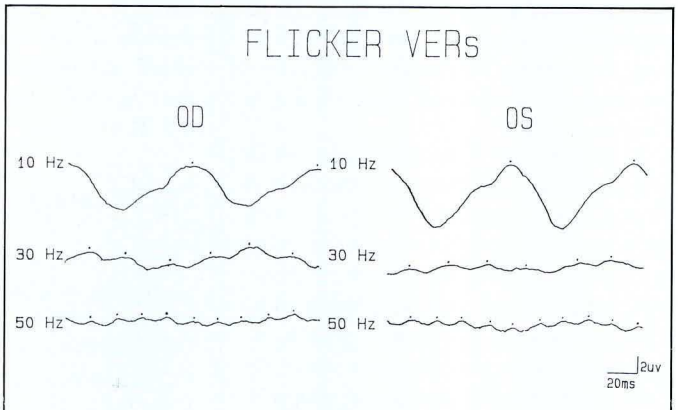


Figure 1
June 1984. Unpatterned VERs for the right and left eye for 10, 30, and 50 Hz flash presentations. No interocular difference was apparent (Bandpass 1-30 Hz, Sensitivity 50μv for all VERs).

constituted a trial. The responses were fed into a Nicolet C.A. 1000 and stored for later analysis.

The unpatterned VERs were of normal amplitude and waveform up to at least 50 Hz flash stimulation (Fig. 1). There were no inter-ocular differences. These findings indicated normal gross functional integrity of the macular-cortical temporal frequency channels.

Patterned VERs were recorded with the same electrode configuration as described for the unpatterned VERs. Steady state patterned VERs were recorded with a T.V. monitor generated 6° checkerboard field that contained black and white checks reversed at 7.5 Hz (space averaged luminance 100 cd/m^2). Thirty 500 ms epochs constituted a trial.

Steady state VERs to 14' arc checks were of normal waveform and amplitude for the unaided left eye. The response was virtually extinguished with the unaided right eye (Fig. 2). The correction of 75% of the myopia (−10 DS) in the right eye produced a recognizable but significantly attenuated response relative to the left eye. With the −10.0 DS lens before the right eye, steady state VERs were recorded to graded check sizes (14', 28', 56' and 112' of arc). The largest amplitude and best waveform was obtained with 56' checks (Fig. 3). This suggested a physiologic acuity potential of at least 6/24 (20/80) with the partial correction. The under correction of the 13 D of myopia by three diopters put the farpoint at approximately 0.33m. With the viewing distance of the checkerboard pattern being 1m, the potential acuity was predicted to be better than the achieved 6/24 acuity if the refractive error were fully corrected. Patient fatigue, however, prevented further assessment.

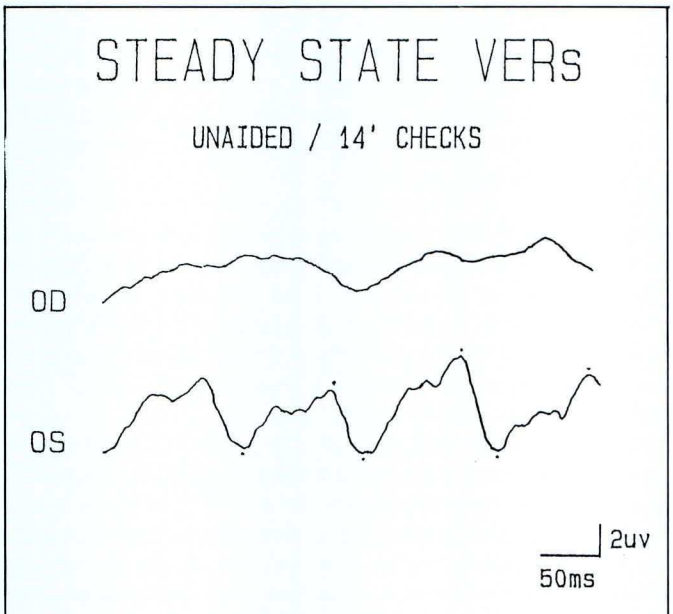


Figure 2
June 1984. Patterned steady state VERs for the unaided right and left eyes, using the 14' arc check size checkerboard.

An external referral was made to confirm the presence of healthy ocular tissues. The ophthalmological assessment supported our findings and revealed no indications of obvious myopic retinal degeneration in the right eye.

In view of the large inter-ocular difference in refractive error, the apparent appreciation of reasonable form vision in the right eye, and the young age of the patient, an internal referral to Contact Lens Services was made. After the successful fit of a contact lens on the right eye, the Electrodiagnostic and Binocular Vision Services would co-ordinate an amblyopia therapy programme.

Treatment

A) Contact Lens Therapy

A decision was made to fit AR's right eye with a soft contact lens using the predicted parameters: 7.8 mm base curve radius (BCR); 12.5 mm diameter; −11.25 D power. These parameters were based on keratometric readings of 45.75 @ 065 and 47.25 @ 175, a spectacle prescription of −13.25 DS and a visible iris diameter (VID) of 11.00 mm.

Since only the B&L lens was available with the desired predicted parameters, a predicted custom lens was calculated. A diameter 1.5 mm to 2.0 mm larger than the VID was chosen so that the lens would not be too large to be inserted by the mother onto her daughter's eye and yet would supply good corneal coverage. Initially the lens was also fitted 0.5 mm flatter than the flattest K reading of the eye to ensure good movement as well as tear and debris exchange. The power was chosen to fully correct the refractive error when vertexed back to the eye. Table 1 indicates the different trial lenses assessed.[12]

TABLE 1
Trial lenses assessed

Manufacturer	Lens	B C R (mm)	Diameter (mm)	Power (D)
ALL Vision	Snowlfex50™	7.8	12.50	−11.25
		7.63	12.8	−13.00
B&L	HO3™	N/A	13.5	−11.00
Cooper	Permalens™	7.7	13.5	−13.50
CCCL	C-Flex70™	7.63	13.0	−13.50
Trans Canada	N & N M79™	7.8	12.5	−11.25
Trans Canada	TC75™*	7.8	12.5	−11.25

*Provided Best Fit

The best tolerated and best fitting initial lens for this patient was the TC-75™ from Trans Canada Optics. Modifications were made to the fitting of the lens after the initial assessment, since all lenses sat at an inferior position and had 2 mm of lag with blinking. Therefore the final lens was made larger in diameter and steeper in base curve. The lens was then better centered and lagged approximately 0.5 mm to 1 mm with the blink. The final lens had the parameters: 7.63 mm BCR; 13.0 mm diameter; and −11.25 D power. A peroxide system was prescribed for daily cleaning and disinfection and a premixed protein cleaning regimen for weekly use.

After three weeks of wear of the contact lens, AR returned to Contact Lens Services for re-evaluation. The lens was comfortable and there were no problems of irritation with the solutions. The patient gradually increased her wearing time to 10 hours/day.

On subsequent check-ups there were no signs of edema or lens intolerance. The over-refraction was found to be +1.00 over the right contact lens. The patient was hyperopic in the left eye (+1.00 −0.25 × 090).

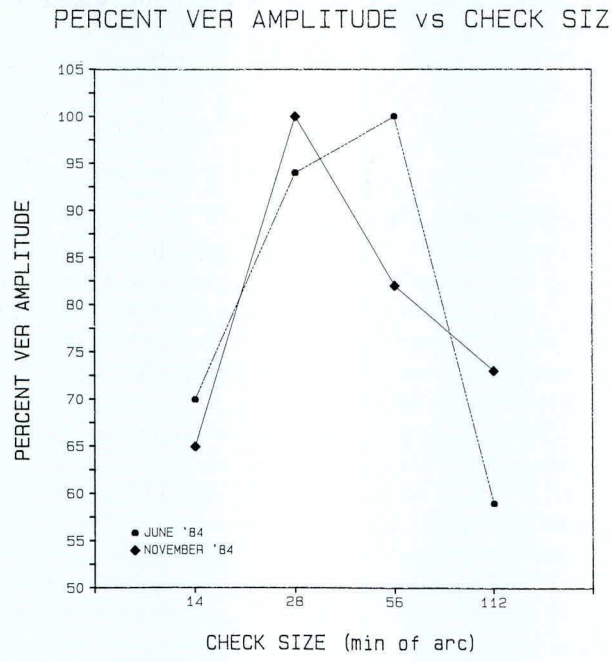


Figure 3

Figure of VER amplitude as a function of check size; plotted as a percentage of the check size producing the largest VER amplitude.

June 1984. The largest amplitude and best waveform was obtained for the 56' arc check size, predicting at least 6/24 acuity.

November 1984. The largest amplitude and best waveform was obtained for the 28' arc check size, predicting at least 6/12 acuity.

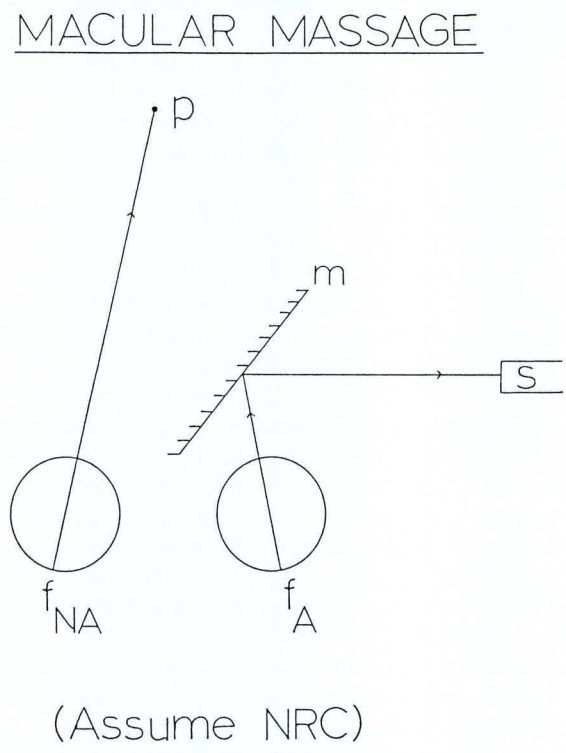


Figure 4A

Schematic for the Macular Massage exercise.
p = fixation point; m = hand held mirror;
s = penlight source; f_{NA} = non-amblyopic left eye fovea;
f_A = amblyopic right eye fovea.
A "game" in which the patient attempts to superimpose the image of the penlight on an indicated fixated target. Performed for about 5 minutes at least 2-3 times per day.

B) Amblyopia Therapy

After the successful fitting of a contact lens on the right eye AR returned to Electrodiagnostic Services in November of 1984 for re-evaluation of the eye's potential physiologic acuity. Figure 3 shows the steady state VERs recorded to 14', 28', 56' and 112' arc checks. The largest amplitude and best waveform was achieved with the 28' arc checks, predicting at least 6/12 (20/40) acuity with the refractive error corrected. This finding, coupled with the apparent cortical differentiation of stimulus size presented to the right eye, suggested a favorable prognosis for amblyopia therapy. The amplitude of the amblyopic right eye VERs was approximately 56% of the amplitude of the non-amblyopic left eye using 28' checks. The subjective acuity using Ffook's symbols was 6/36 (20/120) with the right eye and 6/6 (20/20) with the left eye. Fixation was centred by visualization and 200" of stereopsis was indicated by the Stereofly test.

An amblyopia therapy programme was initiated. Macular massage and the Modified Brock Posture Board were performed daily by the child under the guidance of her mother. Figure 4A and B shows a sketch of the basic design of the Macular Massage and Modified Brock Posture Board exercises that were employed. By January 1985 the subjective acuity had improved enough that macular massage was replaced by daily direct patching. Initially the left eye was occluded with an opaque patch 30 minutes a day. The visual deprivation period was gradually increased to three hours per day. The approach of gradually increasing the patching period was taken to minimize psychological trauma, thereby maximizing patient compliance. The mother was to encourage her child to pursue visually demanding tasks such as colouring, reading, watching T.V. or quiet nearpoint games while patching. As a result of the family's rigorous schedule, the patching and Brock Posture Board programme was not carried out as routinely as had been prescribed. The contact lens was worn everyday.

Steady state VERs performed in May 1985, to 14' checks, confirmed the persisting inter-ocular difference in spatial frequency sensitivity. The right eye amplitude was 35% of the left eye amplitude. The greater inter-ocular difference in amplitude than was found six months earlier was a function of the smaller stimulus check size presented. What was also of interest was the degraded binocular amplitude and waveform. It has been suggested in vision literature that smaller binocular VERs, relative to the constituent monocular VERs, are an objective indicator of binocular dysfunction.[13] AR's relatively attenuated binocular VERs were not a surprise in view of the significant anisometropia and presumed aniseikonia. Over the summer months of 1985 the amblyopia therapy program was suspended due to patient and parent disinterest. The progression in subjective acuity from June 1984 through October 1985 is indicated in Figure 5. As AR's acuity in her right eye improved, our concern over the consequences of aniseikonia increased. Our patient did not verbalize any of the typical symptoms of aniseikonia but her activities were demanding higher levels of binocularity; for

instance playing catch and riding a bike. Consequently, the patient was referred to Aniseikonic Services in June 1985.

C) Aniseikonia Therapy

Aniseikonia was measured using the Multi-meridional Space Eikonometer[14] designed by Arnulf Remole (Fig. 6). The Space Eikonometer consists of one fixed and ten adjustable steel drill rods. The central rod is marked with a red dot for fixation. The other rods are individually brought towards the patient from a position beyond the fixation rod until the patient indicates that the moveable rod is in the apparent frontal parallel plane (AFPP) determined by the fixation rod. The rods can be rotated so as to test for a tilt of the AFPP along with 45°, 135° and 180° meridians.

The absence or correction of aniseikonia results in the AFPP being parallel to the face plane (i.e. a 0° tilt) in all meridians. Aniseikonia manifests itself as a tilt of the AFPP proportional to the magnitude of the size difference. In classic aniseikonia, the tilt of the AFPP is such that the rods closest to the face place are ipsilateral to the eye with largest ocular image.[14] The average size and direction of the AFPP tilt is measured using a protractor indicator placed above the rods.

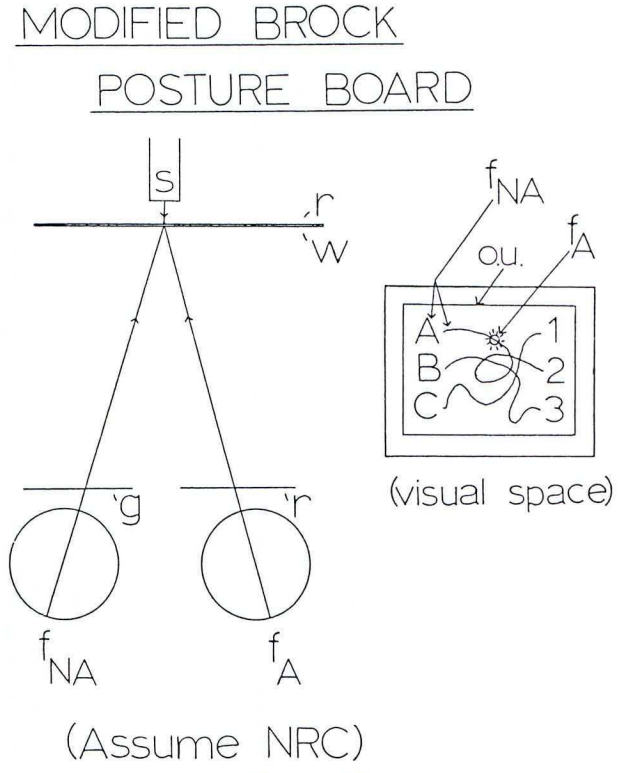


Figure 4B

Schematic for the Modified Brock Posture Board exercise.

s = penlight source; r = red filter; g = green filter

w = white paper, f_{NA} an f_A as in Figure 4A.

The patient attempts to trace a design drawn in *red* pencil on the white paper (seen by the left eye only) with the penlight (seen by the right eye only). Different designs can be drawn on different white sheets of paper to provide variety. A fixation hold, seen by both eyes, is created with a black pencil border around the white paper.

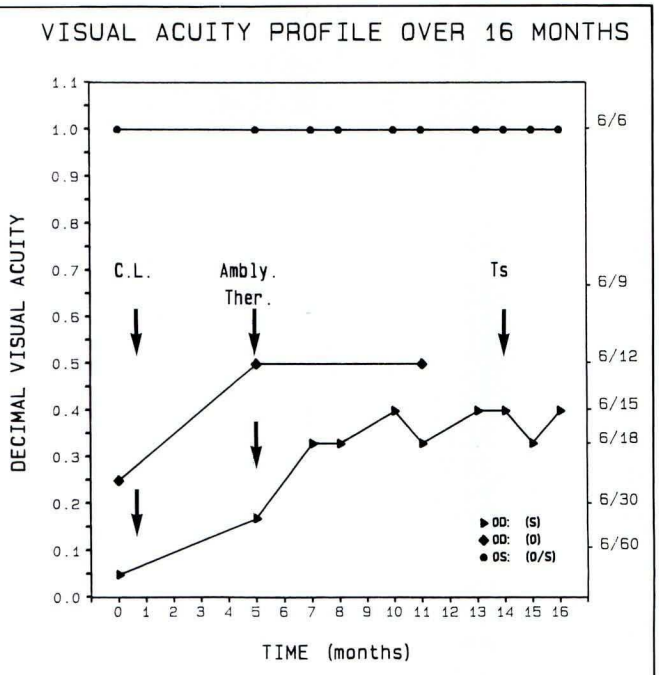


Figure 5

Subjective (S) and Objective (O) visual acuities recorded over a 16 month period from June 1984 through October 1985. The major improvement in subjective acuity occurred in the first seven months and could be attributed to the provision of the contact lens and the initial phases of the amblyopia therapy programme.

(C.L. = the time when the contact lens was first worn on the right eye; Ambly. Ther. = the time when the amblyopia therapy programme was initiated; Ts = the time when the spectacle-contact lens telescope was first worn).

Using the working distance and the patient's interpupillary distance, Ogle developed a mathematical approximation relating the angle of horizontal tilt to the magnitude of aniseikonia.[14,15] The approximation states that a 1% magnification difference corresponds to a tilt of the rods of 3°. An average 12° tilt of AR's AFPP suggested a 4.0% magnification difference. This approximation provided a good prediction since a 4.6% magnifying size lens over AR's right eye virtually eliminated the tilt of the AFPP.

Remole[16] and Enoch[17] described how to incorporate a Galilean telescope to correct aniseikonia in the unilateral aphake. The same principles may be applied to correct an unilateral myope. Before the myopic eye, the telescope is formed by a spectacle-contact lens combination. A plus power spectacle lens forms the "eyepiece" of the telescope and thereby magnifies the ocular image. A contact lens, that is overminused to negate the plus power of the spectacle lens, forms the "objective" of the telescope. The intent of the telescope is to enlarge the image of the more myopic eye so that it equals the image of the less myopic eye.

The calculation of the desired reversed telescopic system for AR was as follows:

1) "Eye piece" Spectacle Lens (F'_Vspec)

$$F'_V\text{spec} = \frac{\Delta\,\text{Mrq}}{\text{t(Mrq)}}$$

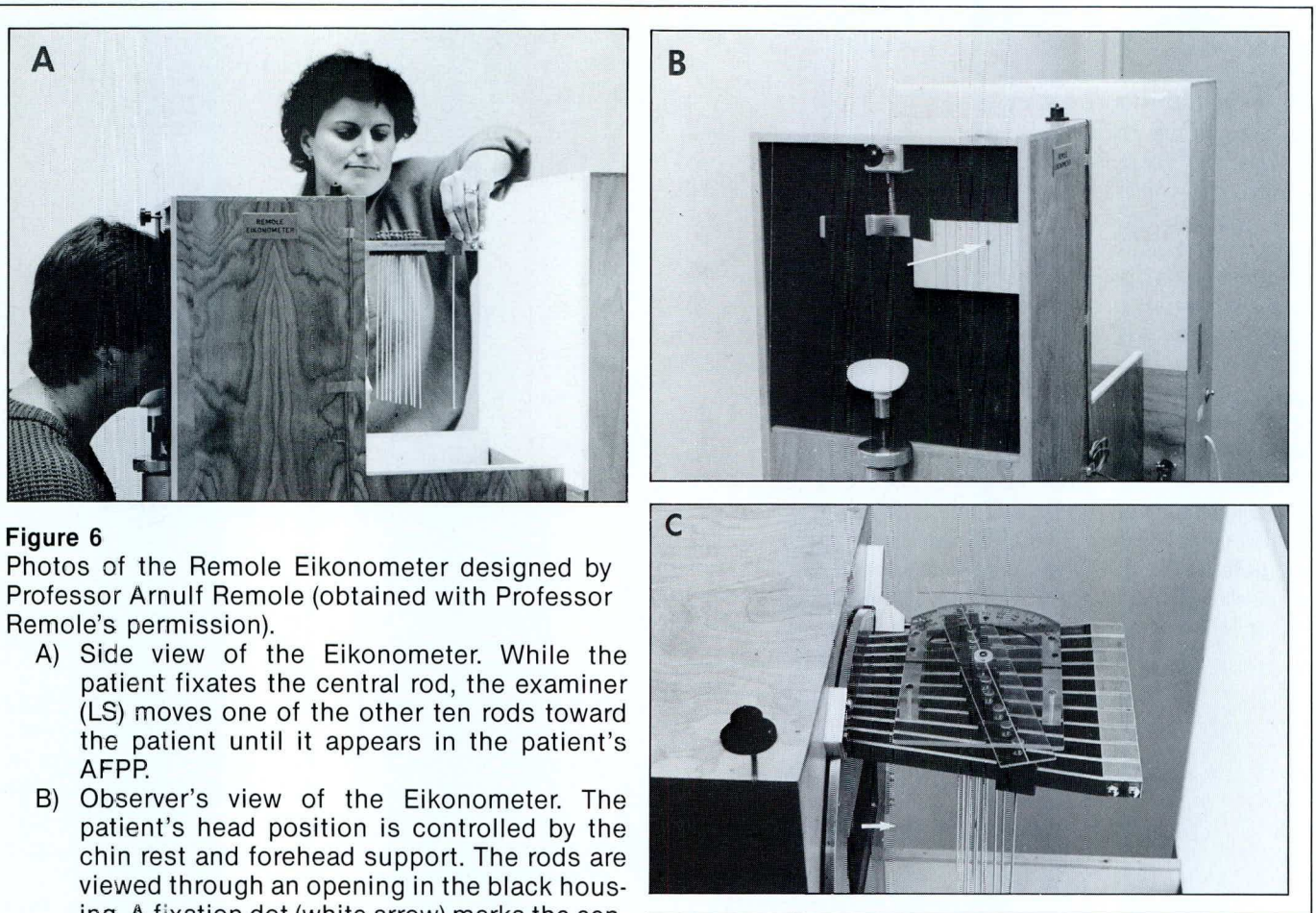


Figure 6
Photos of the Remole Eikonometer designed by Professor Arnulf Remole (obtained with Professor Remole's permission).

A) Side view of the Eikonometer. While the patient fixates the central rod, the examiner (LS) moves one of the other ten rods toward the patient until it appears in the patient's AFPP.

B) Observer's view of the Eikonometer. The patient's head position is controlled by the chin rest and forehead support. The rods are viewed through an opening in the black housing. A fixation dot (white arrow) marks the central rod.

C) Top view of the modified protractor used to measure the tilt of a patient's AFPP. The patient views the rods through the opening (white arrow) in the black housing. This photo illustrates the type of AFPP tilt that AR demonstrated prior to her aniseikonic correction (i.e. the rods on the patient's left side were set closer to the patient than the rods on the right side).

where Mrq = magnification required; t = vertex distance (m)

$$F'_V \text{spec} = \frac{0.046}{.012(1.046)} = +3.66\text{D}$$

$$F'_V \text{spec} \doteq +3.50\text{D}$$

2) "Objective" Contact Lens (F'_{Vcl}) increment

$$F'_{Vcl} = \frac{-F'_V \text{spec}}{1\text{-}tF_V \text{spec}}$$

$$F'_{Vcl} = \frac{-3.50}{1\text{-}0.012(3.50)} = -3.65\text{D}$$

$$F'_{Vcl} \doteq -3.50\text{D}$$

The consistent over-refraction of +1.00D, with the stabilized original contact lens, required consideration in the lens design. The final spectacle-contact lens correction that was provided to compensate for the unilateral myopia and to correct the induced aniseikonia was:

1) Contact Lens Rx:
OD Permalens™: 7.7 mm BCR/13.5 mm diameter
/−13.50 D power
OS none

2) Spectacle Rx:
OD +3.25 DS PD = 50 mm
OS +1.00 −0.25 × 090

A change to Permalens™ was made since when the replacement of the TC-75™ lens was needed the high power was no longer available. The visual acuity subsequent to amblyopia training and spectacle-contact lens therapy was: OD 6/15; OS 6/6

Discussion

Diagnostic Considerations

During a routine oculo-visual assessment, a significant impairment in form perception was uncovered in a healthy three year old fraternal twin's right eye. The diagnosis of anisometropic functional amblyopia affecting the right eye was made after considering the results of a combination of routine and special oculo-visual test procedures.

The diagnosis of a functional amblyopia was contingent upon there being no detectable ocular pathology. The absence of such pathology was supported by both optometric and ophthalmologic assessments of the ocular structures. In addition, objective tests of macular-cortical function were obtained with the VER.

Unpatterned VERs were recorded to ensure that the temporal frequency channels of the visual system were functionally intact and free of detectable pathology. Most vision literature would agree that unpatterned VERs are normal in functional amblyopia.[18-20]

Both waveform and amplitude of patterned VERs are affected by functional amblyopia.[18,21] AR demonstrated the typical reduction in patterned VER amplitude with her amblyopic eye. The cortical differentiation of stimulus size was an encouraging prognostic sign for amblyopia therapy. The significant inter-ocular differences in VER amplitude using small check sizes emphasized the significant disruption of form vision in the amblyopic eye. After the initial VER assessment, periodically recorded patterned VERs provided a useful index by which to objectively monitor neural effects of the therapy programme. The electrophysiologically determined corrected acuity of 6/12 provided a realistic prediction for therapy. After less than one year of a combined contact lens/amblyopia therapy programme, psychophysically determined acuity in the amblyopic eye had reached 6/15.

The diagnosis of anisometropic functional amblyopia was established by the presence of a large uncorrected inter-ocular difference in refractive error in the absence of strabismus, eccentric fixation or pathology.

Therapeutic Considerations

Optometric therapy for this functional amblyope was carried out in three phases: 1) fitting the highly myopic right eye with a suitable contact lens, 2) initiating an amblyopia therapy programme involving a combination of Macular Massage, Modified Brock Posture Board and opaque patching, and 3) correcting aniseikonia with a spectacle-contact lens combination telescope. The first two phases were directed towards maximizing acuity in the amblyopic eye. The final phase was designed to improve binocularity.

The use of a contact lens rather than a spectacle lens was the therapy of choice for two main reasons. Firstly, the contact lens was superior cosmetically. Secondly, the significant interocular difference in image size for this patient was smaller with a contact lens than with a spectacle lens. An important consideration was that the parents were willing to adopt proper care and hygiene in the management of the contact lens. Patient compliance was quite good after the initial contact lens fitting.

Several factors and diagnostic test results were considered to determine the design and prognosis of the amblyopia therapy programme. These included: 1) the patient's age, motivation and health, 2) the type of functional amblyopia, monocular fixation and sensory integration, and 3) the degree of impairment as assessed by both acuity and non-acuity features.

The importance of patient age in determining the prognosis for amblyopia has evolved over the past fifty years. In 1939, McMullen[22] recommended patching and spectacle therapy only for anisometropic amblyopes younger than 10 years of age. Bishop,[10] in 1957, suggested that treatment would be markedly less successful in amblyopes over the age of 11 years; although amblyopes up to 15 years of age might occasionally benefit from amblyopic therapy. Sen[23] supported the treatment of older amblyopic children in a 1982 study of 102 patients. Although the prognosis for improvement of acuity was better in the 6 to 12 year old age group than the 13 to 20 year old age group, 50% of the 46 patients in the latter group showed improved post-treatment acuity. Forty nine percent of the patients showed a concomitant improvement in stereopsis. By all age standards in the vision literature, three year old AR was a desirable candidate for therapy. The fact that AR did not achieve post-treatment acuity better than 6/15 in the amblyopic eye would suggest that factors in addition to age must also be considered.

When dealing with very young patients, parental motivation is as important if not more important than patient motivation. In this case, parental motivation was initially very high. Their motivation did decline as the child reached her acuity plateau.

Oculo-visual assessment revealed a healthy oculo-visual system, free of detectable pathology. ARs general health was excellent. Both of these factors suggested a good prognosis for therapy. Other encouraging prognostic indicators included the presence of centred monocular fixation and gross stereopsis (200").

The type of functional amblyopia may affect the prognosis for therapy. Excluding small angle strabismics, detection and subsequent therapy of strabismic patients often occurs early in life due to their cosmetic presentation (the onset of strabismus also plays a role in the determination of amblyopia of arrest or extinction).[23] In contrast, non-strabismic anisometropic patients are often diagnosed and treated later in life and therefore, may be at a disadvantage with respect to prognosis.[23] AR's parents were predictably surprised at the diagnosis because there had been no indications of visual problems evident in AR's behavior. One must also consider that the degree of amblyopia cannot be directly correlated with the degree of anisometropia. One of the reasons for this is that the magnitude of the anisometropia at the diagnosis may have been different than that which initially existed.[7] The type of unilateral ametropia also affects the prognosis. Unlike an uncorrected significantly hyperopic eye, AR's uncorrected myopic right eye would have had the benefit of some stimulation at near working distances, thereby improving the prognosis for therapy.

When possible, both acuity and non-acuity features should be used to identify amblyopic eyes and determine the therapeutic prognosis.[24-26] Non-acuity amblyopic features include: greater contour interaction, spatial distortion and uncertainty, as well as abnormal pursuit and saccadic tracking.[24,25] Differences in spatial-temporal contrast sensitivities across the visual field also differ between amblyopic and non-amblyopic eyes and even between different forms of amblyopic eyes.[26] Considering the young age of our patient, the easiest and most powerful diagnostic non-acuity tool was the investigation of contour interaction. The effect of contour interaction can be clinically demonstrated by comparing interaction Snellen and interaction-free tumbling E acuities. A functional amblyope will usually perform inferiorly when the acuity test includes interaction. An encouraging prognostic sign for amblyopia therapy is an improvement in acuity demonstrated when contour interaction is eliminated. For AR, her initial corrected interaction-free acuity (IFA) was 6/30.

Within two months of wearing the right contact lens daily and performing amblyopic exercises, the IFA was 6/21. Six months later, after continuing amblyopic exercises intermittently, the IFA plateaued at 6/15. Interaction acuities using conventional Snellen optotypes were always unsuccessful despite the child being lettered. This might have been an indication of the significant effect of contour interaction, spatial distortion and uncertainty. In part, the reduction in acuity demonstrated with the Snellen chart may have been the result of the higher level of patient co-operation required; specifically the need for vocalization of the target identity. The IFA was obtainable without vocalization because the child used hand symbols to represent what she saw. Shyness was

not considered to be a major factor since the child became quite familiar and comfortable with the clinicians over the numerous visits.

The amblyopia therapy programme was designed keeping the patient's age and level of amblyopia in mind. Exercises were chosen that minimized demands on patient effort and attention span yet maximized the development of visual function. Patching was initiated only after acuity had improved using a combination of the Macular Massage and Modified Brock Posture Board exercises. This strategy was adopted to reduce any potential hazards created by AR relying solely on a highly amblyopic eye for mobility and subsequently maximize patient compliance during patching. Compliance was also enhanced by one of the parents handmaking a patch that was more cosmetically acceptable yet still met our therapeutic specifications. During periods of patching, visually stimulating activities were encouraged such as: colouring, tracing and drawing. As is frequently the case, patient and parent motivation was directly related to the degree of perceived visual improvement.

The aniseikonic therapy was initiated out of a concern for binocular function. The provision of spectacle-contact lens combination telescope before the right eye and a spectacle lens before the left eye provided an optical correction that minimized interocular differences in the stimulus to accommodation and ocular image size.

Unfortunately, AR was discharged from the School of Optometry prior to electrophysiologically studying the efficacy of the aniseikonic correction. AR's care was transferred to an optometrist in private practice whose location and hours were more convenient for the family. One year after discharge from the School, AR was still wearing her contact lens and spectacles. Her acuities were unchanged and there were behavioral indications of improved binocularity.

This case helps emphasize the importance of both the early detection of significant anisometropia and the subsequent tailoring of a therapy programme appropriate for the particular patient. The use of the VER both as an objective diagnostic tool as well as an objective prognostic tool is illustrated. This report also demonstrates the relative ease of fitting a contact lens of high prescription, provided a local custom lab is available to manufacture the appropriate lens. Finally, the need for multi-phased therapy in some patients to attend to both monocular as well as binocular functional integrity is addressed by the employment of aniseikonic therapy subsequent to refractive and amblyopic therapy.

Acknowledgements

Thanks to Drs. Barbara Drader, Dagmar Lutzi and Arnulf Remole for their dedicated optometric services, Lynda Lang and Jay Van Laar for their secretarial support, as well as Karen Ballard and Manfred Rieck for their photographic work. MM Spafford was supported by a 1986 COETF grant.

References

1. Duke-Elder S. System of Ophthalmology. Ocular Motility and Strabismus. London: Kimpton, 1973; VI: 296.
2. Sullivan M. Results in the treatment of anisometropic amblyopia. *Amer Orthopt J* 1976; 26: 37–42.
3. Schapero M. et al. Dictionary of Visual Science. 2nd Ed. Pennsylvania, Chilton, 1968; 23–24.
4. von Noorden G.K. Binocular Vision and Ocular Motility. Theory and Management of Strabismus. 3rd Ed. St. Louis Mosby, 1985; 210–214.
5. Levi D.M., Harwerth R.S. A sensory mechanism for amblyopia: Electrophysiological studies. *Am J Optom Physiol Optics* 1978; 55(3): 163–171.
6. von Noorden G.K. Classification of amblyopia. *Am J Ophthalmol* 1967; 63(2): 238–244.
7. Cibis L.M. Treatment of Amblyopia In: Becker B., Burde R.M. eds. Current Concepts in Ophthalmology. St. Louis, Mosby 1969; 2: 213–225.
8. von Noorden G.K. Experimental amblyopia in monkeys. Further behavioral observations and clinical correlations. *Invest Ophthalmol* 1973; 12(10): 721–726.
9. Hubel D.H., Wiesel T.N. The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *J Physiol* 1970; 206: 419–36.
10. Bishop J.W. Treatment of amblyopia secondary to anisometropia. *Brit Orthoptic J* 1957; 14: 68–74.
11. Odom J.V., Hoyt C.S., Marg E. Eye patching and visual evoked potential acuity in children four months to eight years old. *Am J Optom Physiol Optics* 1982; 59(9): 706–717.
12. Sorbara L., Callender M. et al. Hydrogel lenses available in Canada update. University of Waterloo, School of Optometry, *Contact Lens J* 1985; XII(1).
13. Srebro R. The visually evoked response. Binocular facilitation and failure when binocular vision is disturbed. *Arch Ophthalmol* 1978; 96: 839–844.
14. Remole A. A New Eikonometer: The multimeridonal apparent frontoparallel plane. *Am J. Physiol Optics* 1983; 60(6): 519–529.
15. Ogle K.N. Analytical treatment of the longitudinal horopter: Its measurement and application to related phenomena, especially to the relative size and shape of the ocular images. *J Opt Soc Am* 1932; 22: 665–728.
16. Remole A. Prismatic effects of iseikonic spectacle-contact lens telescopic systems. *Can J Optom* 1984; 46(3): 122–128.
17. Enoch J.M. A spectacle-contact lens combination used as a reversed Galilean telescope in unilateral aphakia. *Am J Optom* 1968; 45: 231–240.
18. Levi D.M. Patterned and unpatterned visual evoked responses in strabismic and anisometropic amblyopia. *Am J Optom Physiol Optics* 1975; 52: 455–464.
19. Fishman R.S., Copenhaver R.M. Macular disease and amblyopia; the visual evoked response. *Arch Ophthalmol* 1967; 77(6): 718–725.
20. Nawratski I., Averbach E., Rowe H. Amblyopia ex anopsia; the electrical response in retina and occipital cortex following photic stimulation of normal and amblyopic eyes. *Am J Opthalmol* 1966; 61(3): 430–435.
21. Sokol S., Nadler D. Simultaneous electroretinograms and visually evoked potentials from adult amblyopes in response to a pattern stimulus. *Invest Ophthalmol Visual Science* 1979; 18(8): 848–855.
22. McMullen W.H. Some points in anisometropia. *Trans Ophthalmol Soc UK* 1939; 59; 119–24.
23. Sen D.K. Results of treatment of anisohypermetropic amblyopia without strabismus. *Brit J Ophthalmol* 1982; 66: 680–84.
24. Flom M.C., Bedell H.E. Identifying amblyopia using associated conditions, acuity, and nonacuity features. *Am J Optom Physiol Optics* 1985; 62(3): 153–160.
25. Levi D.M., Klein S.A. Vernier acuity, crowding and amblyopia. *Vision Res* 1985; 25(7): 979–991.
26. Hess R.F., Pointer J.S. Differences in the neural basis of human amblyopia: The distribution of the anomaly across the visual field. *Vision Res* 1985; 25(1): 1577–1594.

VISION CARE NEWS/ACTUALITÉ OCULO-VISUELLE

IOOL News Ranges From Great to Gloomy

Dr. Roland C. des Groseilliers, a past President of CAO, is currently Vice President of the International Optometric and Optical League. Recently, he provided us with a variety of news items that indicate once more just how fortunate the optometric situation is in North America when compared with its dilemmas elsewhere.

(1) Optometry in Recession

- England's House of Commons, as of this writing, has given second reading to a bill that totally eliminates "sight testing" from that country's National Health insurance program.
- Australia has decided that its own health care plan will now fund eye care to the tune of one examination every two years, a change from the previous arrangement which covered an annual examination.
- Under a new law in France, optometrists there can now fit contact lenses only on patients referred to them by an ophthalmologist.
- And in Greece, if you practice Optometry, you are guilty of a crime — the profession has been declared illegal.

(2) Opening the Eyes of the World

In response to what it correctly perceives as a growing professional crisis, the IOOL has launched an international campaign for members and for support of a number of worthwhile objectives:

- responding to the increasing numbers of unqualified sellers of optical products, "off-the-shelf" spectacles and fitting contact lenses without following thorough examination procedures;
- encouraging national governments to reverse anti-optometric legislation for the sake of public protection;
- supporting international efforts to establish and develop Optometry.

The IOOL fears that, if current statistics are projected through one more generation, the world will contain over 100 million blind people. Restrictive legislation, they maintain, is preventing the early detection and treatment that could prevent many of these incidences.

(3) Patron? International Patron?

As part of its campaign, the IOOL has established two new categories of "closely involved" membership, noted above. A £500 donation to the League earns the International Patron designation, while a Patron is an individual who donates £250. Both donations will be acknowledged by a special certificate signed personally by Dr. G. Burtt Holmes, IOOL President.

Payments can be made (i) as a one-time donation or, in the case of the International Patron donation, split over two payments in the course of a year; (ii) via VISA, Master Card, Access Card or Barclaycard.

The IOOL is a federation of 56 national optometric associations all with the common goal of fostering and encouraging optometric growth and development.

For further information (Remember, however, to send any cash or cheque **donations** to **CAO in Ottawa** to ensure your receipt of tax deductibility. We will ensure the full amount is then forwarded directly to the League.):

The International Optometric and Optical League
10 Knaresborough Place
London, SW5 0TG
ENGLAND

(4) Convention Preview

The 1989 Annual Meeting of the IOOL will be held in Luxembourg.

(5) World Optometric Education Needs Help

The IOOL is also seeking any used vision and eye care textbooks for use in optometric training institutions around the world.

In addition, on World Optometry Day, the League is asking anyone and everyone to donate old spectacles for distribution to Third World patients.

(6) Corporate Donation to Promote IOOL in US

Bausch and Lomb recently gave a grant of $12,000.00 to the American Optometric Association, of which current IOOL President Dr. G. Burtt Holmes is a member, for purposes of promoting the League to the AOA membership.

Alberta Optometric Advocates

Elsewhere in this issue, CAO President Dr. Scott Brisbin, in his *President's Podium*, announces the formation of the Alberta Chapter of the CAO Optometric Advocates, although still to be constituted formally as a national Section.

Shown here following the Optometric Advocates' inaugural meeting are: (L-R) from the newly elected Executive: Cathy Dawdy (Secretary Treasurer); Anita Patel (Vice President); Calli Brisbin (President) and special guest Alana LaRock, President of the American Optometric Association Auxiliary.

CAO members interested in pursuing the establishment of an Advocates' group in their own province are invited to contact Dr. Brisbin directly.

VISION CARE NEWS/ACTUALITÉ OCULO-VISUELLE

Sola Ophthalmics Wins Patent Infringement Judgement

As a result of a recent ruling by the US District Court in Phoenix, Arizona, two companies, Paragon Optical and Wilsa Incorporated have been judged to have infringed on a number of gas permeable contact lens patents held by Sola Ophthalmics.

Specifically, Paragon's Paraperm O_2, O_2 Plus and EW contact lenses and Wilsa's Optacryl 60, K Ext and Z Contact Lenses and lens materials were ruled to have infringed on Sola's US patents for its own line of gas permeable lenses.

The presiding judge also awarded damages to Sola of $18.1 million through 1985, which he then doubled to $36.2 million in determining that the infringement had been willful.

Damages for the years 1986 and 1987 have yet to be determined.

—From a Sola Ophthalmics news release received at CAO November 9, 1987

Lens Opacity Measuring Instrument from IntraOptics

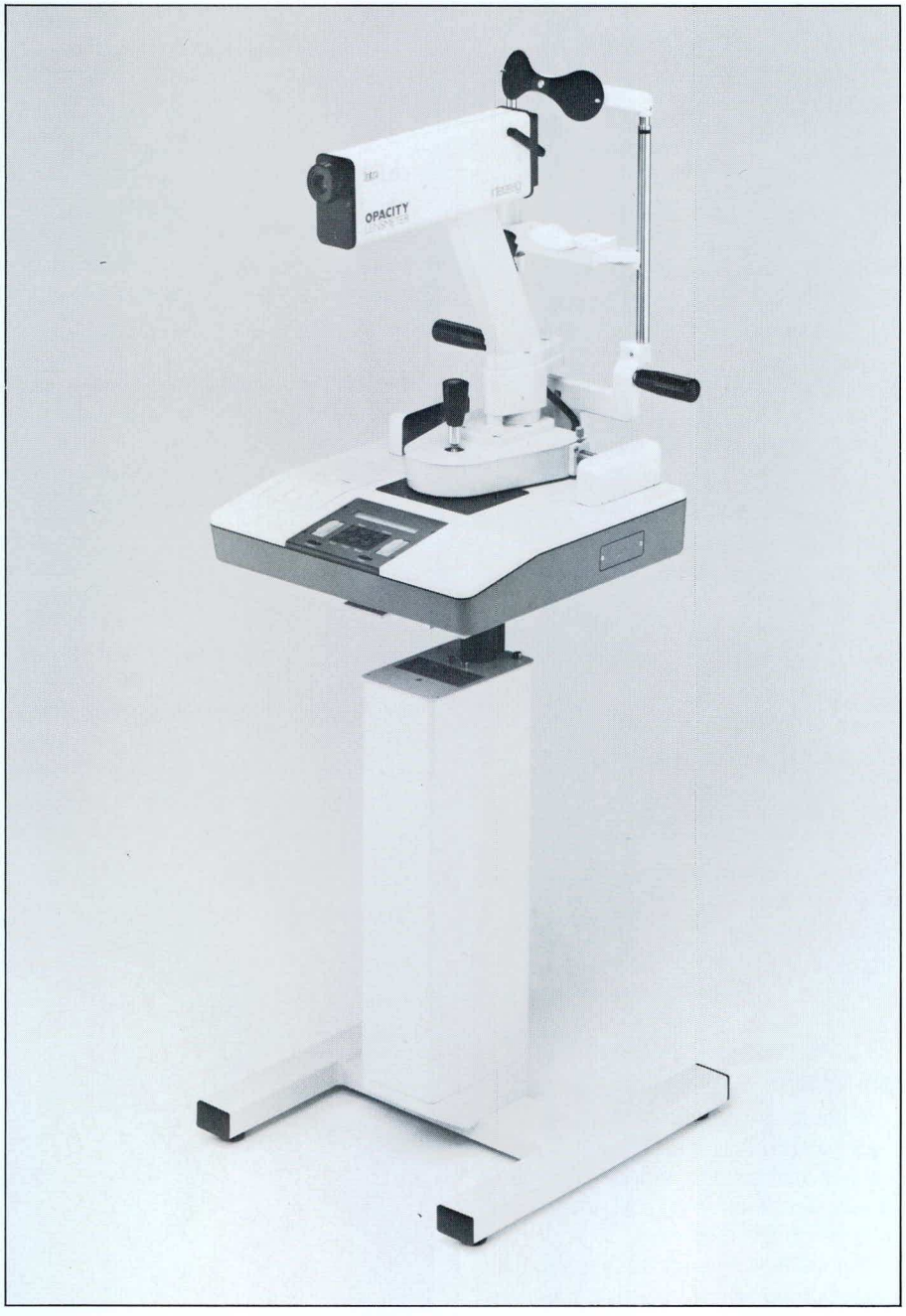
A new instrument from IntraOptics, Inc. is being introduced as "the first instrument to objectively measure and document the degree of opacity in the human eye".

The Opacity Lensmeter is designed to enable practitioners to track cataract development through accurate, quantitative measurement of opacity, and to produce a printed record (a digital printout based on a calibrated opacity scale of 1–99) of the measurements.

IntraOptics' President, James R. Cook, M.D., said in a recent news release that the instrument can be used to detect a cataract condition even in its early stages of development.

The Opacity Lensmeter was developed originally to enable monitoring of the lenticular effects of topical, oral or injectable steroids, some of which have been suspected of causing cataracts or glaucoma.

Information:
Peter Molinaro
IntraOptics, Inc.
PO Box 317
Huntington, West Virginia
25708, USA
Tel. (304)528-2000



KW Optical Consulting Services helping optometrists in Practice Management

In this issue, *CJO * RCO* readers will note the return of KW Optical to our advertising pages (inside front and back covers).

KW has unveiled a new corporate arm — KW Optical Consulting Services.

According to a company representative, more than 50 years of providing services to optometric clients and students has revealed a need for consultation in the many aspects of running a successful practice. Accordingly, KW Optical Consulting Services has been established as a division of the company. Acting as a confidential referral service, KW Optical

VISION CARE NEWS/ACTUALITÉ OCULO-VISUELLE

Consulting Services is a source which both new and existing optometrists may use to obtain advice from professionals and other qualified individuals in the following areas:

ACCOUNTING:
- development of accounting systems
- preparation of financial statements
- preparation of tax returns
- development of cash flow forecasts and financing proposals

BANKING:
- daily banking requirements
- professional finance programs including current operating loans and term financing for practice acquisition, equipment purchases, leasehold improvements, etc.

COMPUTERIZATION:
- evaluation of need for computerization of your practice
- integrated practice management system developed for Optometrists
- accounting and word processing software
- Rx submission via telecommunications

INSURANCE:
- income protection plans
- life insurance
- practice liability insurance
- general insurance
- staff benefit programs including health and disability protection

LEGAL:
- review of leases and real estate agreements
- associate, partnership and shared cost agreements
- practice liability counselling

OFFICE DESIGN:
- evaluation of office design alternatives
- development of practice layout plans
- renovations and new construction

PRACTICE MANAGEMENT:
- reviews of staff compensation and benefits
- demographic studies
- human resources consulting
- marketing your practice

PRACTICE PLACEMENT:
- practice evaluations
- identification of start up locations
- associate and partnership referrals

KW Optical Consulting Services has assembled this group of respected individuals and organizations to help you in all areas of practice management. If you require assistance or desire further information please contact:
John D. Uhrig, Brent Heard, Siegfried Wolf at (519)743-2601

Coming Events

(**NOTE:** For further information on an event sponsored by the Canadian Association of Optometrists, contact the CAO office in Ottawa. For an event sponsored by a provincial Association or Society of Optometrists, contact the respective hosting organization. The address for an international or outside Canada event is included with the item.)

1988

MARCH/MARS

6: **Canadian Association of Optometrists Association Canadienne des Optométristes Dial-the-President 1988 Ligne Direct au Président 1988**
Ottawa, ON

6 - 12: **Save Your Vision Week in Canada Semaine de la Vision au Canada "Life Is Worth Seeing!/La Vie, Faut Voir Ca!" Honorary Chairperson — Wayne Gretzky — Président d'honneur**

APRIL/AVRIL

20 - 22: **Ontario Association of Optometrists**
Congress '88
Holiday Inn Airport
Toronto, ON

21 - 27: **5th Australian — International Optometric Congress**
Manly - Pacific International Hotel
Sydney, Australia
Information:
Australian Optometrical Association
204 Drummond Street
Carlton, Victoria, 3053 Australia
Tel.(03)663-6833

23: **Saskatchewan Association of Optometrists**
Council Meeting
Regina, SK

23 - 24: **Manitoba Optometric Society**
Annual General Meeting
Winnipeg, MB

30 - May 3: **Optica '88 World Optical Trade Fair**
Cologne, West Germany
Information:
KolnMesse
c/o Canadian German Chamber of Industry and Commerce Inc.
Suite 1410
480 University Avenue
Toronto, ON
M5G 1V2
Tel.(416)598-3343

30 - October 30: **Expo Down Under (World Expo 88)**
Brisbane, Australia
Information:
The Communications Division
World Expo 88
234 Grey Street
PO Box 1988
South Bank,
South Brisbane
Queensland, 4101
Australia
Tel. 07-840-1988

MAY/MAI

6 - 7: **Atlantic Association of Optometrists**
Continuing Education Program

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Keddy's Brunswick Inn
Moncton, NB

7: **British Columbia Association of Optometrists**
Council Meeting
Victoria, BC

8: **British Columbia Association of Optometrists**
Murder Mystery Day
In Conjunction With 1988 Annual General Meeting
Victoria, BC

9 - 10: **British Columbia Association of Optometrists**
Annual General Meeting
Victoria, BC

11 - 14: **Saskatchewan Association of Optometrists**
Continuing Education Program
Saskatoon, SK

12: **Saskatchewan Association of Optometrists**
Special General Meeting
Saskatoon, SK

25 - 28: **International Contact Lens Centenary Congress (ICLCC '88)**
Queen Elizabeth II Conference Centre
Westminster,
London, England
Information:
ICLCC '88
Conference Associates
27A Medway Street
London, SWIP 2BD UK
Tel.(01)222-9493

JUNE/JUIN

4 - 6: **Association des Optométristes du Québec**
11e Symposium annuel des optométristes (4 juin)
Auberge Estrimont, Qué

Assemblée générale annuelle (5 juin)
Orford, Qué

Tournoi de golf (6 juin)
Orford, Qué

7 - 11: **Alberta Association of Optometrists**
Optometric Practice Development Week (Seminar)
Calgary Convention Centre
Calgary, AL

AUGUST/AOUT

27: **Saskatchewan Association of Optometrists**
Council Meeting
Saskatoon, SK

SEPTEMBER/SEPTEMBRE

TBA **Saskatchewan Association of Optometrists**
5th Annual Sand Pail Golf Tournament
Yorkton, SK

15 - 17: **New Brunswick Association of Optometrists**
Keddy's Brunswick Inn
Moncton, NB

23 - 24: **Newfoundland Association of Optometrists**
Annual General Meeting
Gander, NF

24 - 25: **Alberta Association of Optometrists**
Annual General Meeting
Grande Prairie, AL

OCTOBER/OCTOBRE

7: **Association des Optométristes du Québec**
Réunions annuelles de la L.S.O.
Hotel Reine Elizabeth
Montréal, Qué

8 - 9: **Association des Optométristes du Québec**
10e Colloque International sur la Lentille de Contact
Hotel Reine Elizabeth
Montréal, Qué

TBA: **Manitoba Optometric Society**
Semi-Annual Meeting

TBA: **Nova Scotia Association of Optometrists**
Annual General Meeting
Halifax, NS

NOVEMBER/NOVEMBRE

TBA: **Prince Edward Island Association of Optometrists**
Annual General Meeting

3 - 6: **Saskatchewan Association of Optometrists**
Council Meeting
Regina, SK

4 - 5: **Saskatchewan Association of Optometrists**
79th Annual General Meeting
Regina, SK

4 - 7: **5th International Retinitis Pigmentosa Congress**
Hyatt Regency Hotel, Melbourne, Australia
Information:
RP Australia Conference Secretary
46A Oxley Road
Hawthorn 3122
Victoria, Australia
Tel.(03)819-6590

The Canadian Journal of Optometry
la Revue Canadienne d'Optométrie — Annual Index Volume 49 — 1987

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ARTICLES

EDITORIALS

BOOK REVIEWS

FEATURES / ANNOUNCEMENTS

LETTERS/COURRIER

Editor, *CJO * RCO*

I enjoy reading *The Canadian Journal of Optometry * la Revue Canadienne d'Optométrie* but feel a few changes or additions would make it even more enjoyable.

I believe optometrists across the country should be aware of changes that are occurring in the profession in each province. Practising in Ontario, I know nothing of the issues affecting other OD's, for example, in Alberta or British Columbia. Problems arising in one province can, in the future, affect others. Our national Association Journal, therefore, should have a survey of provincial matters. This will supplement the scientific and research works that, of course, are necessary in such a publication.

In addition, however, I feel that more Editorials or Guest Editorials should be included. Optometrists must have a forum to express their concerns, ideas and opinions. Presently, for example, the President's Podium editorials have been excellent. But other prominent and not so prominent OD's should also have a chance to share their wisdom or thoughts on particular topics.

I know that these suggested changes would be welcomed not only by myself, but also by other OD's to whom I have talked.

This is a Journal for optometrists, so let it reflect our moods, our thoughts, our good and bad points.

Following are some topics that I feel this profession must face in the future:

Optometry, to my mind, is just entering a new era. Our profession is naturally evolving, but a new awareness is just beginning to emerge.

We no longer are just "refractionists" or "contact lens fitters" but, every so slowly, we are being used by our patients as consultants and, yes, as providers of primary eye care. To strengthen this role, I feel that the following changes should occur to add to the foundation that has been formed in the past:

1) **Drugs** Optometry requires the use of pharmaceutical drugs, diagnostics (as most provinces have) and, in the future, therapeutics. Without their use, we limit our ability to provide total care. The benefits from their use can far outweigh any risks than might be present. As most optometrists become comfortable using diagnostic drugs, therapeutics will be the natural extension. Optometrists are much more accessible to the public and we are better trained to treat eye conditions than the general practitioner. An increased use of, particularly, therapeutic drugs will also elevate the profile of our profession in the eyes of government and other health groups. Dentists presently have the use of therapeutic drugs, even though they do not have "medical" training. Also, by the end of 1987, over half the states in the U.S.A. had the use of therapeutic drugs. Optometry in our country should also progress towards this broadening of care; we have the skills.
2) **Training** To move us further into primary care, optometry students in particular require clinical, hands-on experience. While schools presently provide needed exposure to patients, we should have access to other teaching facilities, such as hospitals. Here, our students can not only observe and participate, but learn, learn and learn. These publicly-funded institutions should be opened up to allied health providers. Physicians must not monopolize these facilities. If an interdisciplinary approach is to work, changes in the health field must occur here. Optometry must not be isolated, but should cultivate relationships with other professions and grow as members of the health community.
3) **Government** Governments increasingly control how health care dollars are spent. We must make sure that we are a dominant force in the vision and eye care field and that our services are adequately compensated. We know we provide excellent care, but we must demonstrate to the government the full scope of our talents so we can assume the role of primary vision and eye care providers. Further, we must not allow ourselves to be intimidated by any group opposing our goals.
4) **Vigilance** Optometry must be highly vigilant and united. We must constantly refine our skills and master new ones, so we will always be progressive. We must ensure that our profession is not dominated by business corporate interests that see our skills as a means solely to making profits. We can look to the U.S.A. to see how corporations have made many O.D.'s mere employees and, yes, just refractionists again. If we do not guard against outside interests, independant optometrists one day may be as scarce as independant pharmacists. Delivery of health care services will be an important budget issue in the near future. We must guarantee that our services are not restricted and that freedom is not lost for our patients.

   Dentists presently are facing the threat of capitation and are experiencing the effects of an outside interest on their profession. Their unity will be put to the test.

   To keep strong, we must maintain high standards so our patients obtain complete, competent care. It is up to each practitioner to provide the best care to each patient each day. Only in this manner can we assume this important role of primary care, and only in this manner do we deserve it. By providing this care, we will always have the public's support and their respect.

Dr. E.A. Pidutti
Optometrist
Sudbury, Ontario

BOOK REVIEWS

Dictionary of Optometry: ***Michel Millodot, O.D., Ph.D., F.B.C.O., F.A.A.O. Butterworth's, London, 1986. 190 pp, over 2,500 terms. £12.50***

This small dictionary must be considered as a companion volume to the same author's ***Dictionnaire de la Science de la Vision***, which has a multidisciplinary objective. This second work is directed to the Profession of Optometry and contains a far greater number of terms. It is solely in English while the first work is in French with an English Index appended.

The reader should not be misled by the title into thinking that the book is restricted exclusively to "optometric" terminology. It is not. It covers all disciplines which are in one way or another directly or indirectly related to optometric training and practice. Thus, terms relevant to Optometry, Optics, Lenses, Physiological Optics, Psychology, Anatomy, Neurology, Pharmacology, Instrumentation and equipment, Ergonomics, Systemic and Ocular Disease will be found in their appropriate alphabetical order.

Not only are basic terms well and clearly defined, but synonyms are included as well, with cross referencing indicated. To further facilitate retrieval, there are long lists of specific examples under general headings. For example, following a brief definition of the word, "theory", there are some 30 specific examples, from "Bielchowsky's Theory" to "Zeeman's Theory"; 24 under "corneal" ("Corneal bedewing" to "corneal ulcer") and some 20 under "disease" ("Basedow's Disease" to "Tay-Sachs Disease").

The book is paperbacked with a stiff, glossy cover. The text is well printed on a good quality, matt finished paper. At 250 gm and 21.6 × 13.8 cm, it is neither heavy nor cumbersome. Students and office assistants should find it a useful reference. Practitioners will find it not only a Dictionary, but a useful text for Continuing Education or as a memory freshener.

Its modest cost makes it accessible to all, particularly students.

GMB

The Canadian Journal of Optometry
la Revue Canadienne d'Optométrie

List of Reviewers

The Editors of the *CJO * RCO* would like to extend their thanks and appreciation to the following individuals who have consented to review papers submitted for publication.

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University of Waterloo

PRESIDENT'S PODIUM/MOT DU PRÉSIDENT

(con't from page 170)

An Optometric Advocates Section is composed of non-optometrists who believe in the vital role optometrists play on the primary health care team and whose members have as an objective the sharing of what they know of the value of Optometry with others.

Many, for example, will have stronger and more effective political connections than most members of our own optometric Keypersons Committee. Many will have better communication links with the education system, with other health care professions, etc. At some level or other, they will all be messengers.

The potential for such a group is exciting. Getting it organized in each province will be a challenge. I encourage you to talk it up amongst your families, your staff, your patients and your friends. Make a list of potential messengers for Optometry that you know. Then call an organizational meeting (perhaps, but not necessarily, in conjunction with your next provincial AGM).

I expect, in fact, that this will be one subject about which I will be asked on Sunday, March 6 for **Dial the President Day**. Call me and let's discuss it further. Then get yourself and all those other potential advocates involved.

Your future may depend on it.

Scott D. Brisbin, O.D., F.A.A.O., President

(suite de la page 169)

trois "O": l'optométriste, l'ophtalmologiste et l'opticien.

La majorité des répondants ont même classé les opticiens (et les distributeurs de produits ophtalmiques) au-dessus des optométristes et des ophtalmologistes comme sources les mieux informées à consulter en cas de problèmes visuels! (Cependant, Tom Sullivan prétend que je ne devrais pas me surprendre de cette situation.)

Nous devons transmettre le message, et 2 600 optométristes au Canada ne suffisent pas à cette tâche. Alors qui va nous prêter main-forte?

La minorité qui connaît le rôle important de l'optométrie dans le système des soins de santé — voilà la tranche de la population qui peut nous aider. Ce sont les familles des optométristes, l'équipe du personnel auxiliaire qui dispense chaque jour ses précieux services, les représentants des sociétés ophtalmiques qui ont pris le temps de connaître leurs clients et leur travail, les "bons" patients, les professeurs, entraîneurs et autres travailleurs du domaine de la santé qui ont eu l'occasion d'observer les bienfaits de soins optométriques ou d'en être eux-mêmes les bénéficiaires...

Eux preuvent nous aider à diffuser le message.

Ce sont eux qui peuvent grossir de dix fois le nombre de nos messagers. Ils représentent une ressource inexploitée à découvrir et à mobiliser si l'on veut que le message de l'optométrie gagne une part importante de la grande majorité.

Je suis heureux d'affirmer que cette idée commence déjà à germer en Alberta. L'assemblée générale annuelle 1987 de l'Association des optométristes de l'Alberta, tenue à Calgary en novembre dernier, a vu naître le chapitre albertain de la Section

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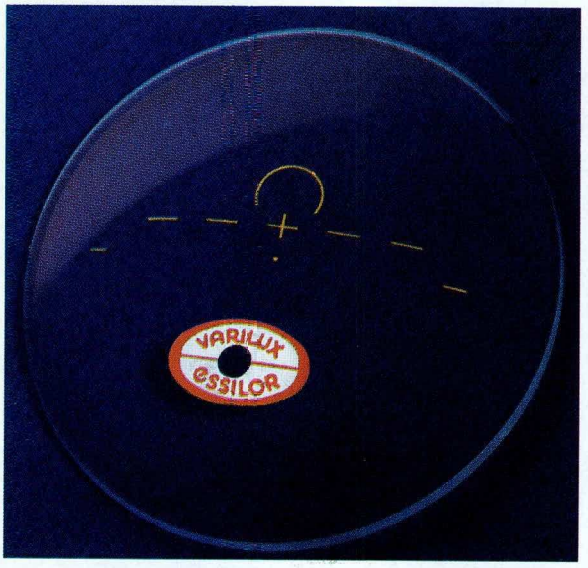

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MOT DU PRÉSIDENT

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de promotion de l'optométrie de l'ACO. J'espère que cette tendance se poursuivra par la création d'autres chapitres dans les provinces, de la Colombie-Britannique jusqu'à Terre-Neuve.

L'Alberta a eu l'honneur d'accueillir à sa réunion inaugurale la présidente de l'American Optometric Association's Auxiliary, Alana LaRock, qui a prodigué ses encouragements et conseils judicieux. On y a discuté entre autres d'exemples précis de projets de diffusion du message de l'optométrie qui ont été menés à bonne fin, du nouvel équipement audio-visuel excellent qui est de plus en plus répandu, de lignes directrices d'organisation, etc.

Une branche auxiliaire, telle l'AOA, peut se révéler une aide inestimable pour l'optométrie. Mais elle souffre d'un malaise étouffant: elle ne regroupe que les conjoints et élimine de ce fait nombre de nos messagers les plus précieux.

Il y a là problème d'image, éprouvé même par les conjoints des optométristes. Puisque de plus en plus de couples sont tous deux professionnels et que de plus en plus de femmes intègrent la profession de l'optométrie, l'idée de se joindre à un groupe d'auxiliaires, qui évoque à tort des "cercle de couture", n'est pas très alléchante pour nombre de conjoints d'optométristes, hommes ou femmes.

Voilà pourquoi la notion d'auxiliaire a été rejetée depuis longtemps dans la plupart des régions du pays, et le groupe de l'Alberta s'est fait redire maintes et maintes fois qu'il n'était pas un groupe d'auxiliaires.

Nous avons l'occasion de recommencer à neuf, d'éviter les écueils qui entourent tout groupe d'auxiliaires, mais surtout de construire sur les solides assises de l'AOA et d'autres associations semblables afin de créer le service de messagers le plus efficace qui soit.

La Section de promotion de l'optométrie se compose de non-optométristes qui croient au rôle vital des optométristes au sein de l'équipe des soins primaires, et qui se fixent comme objectif de partager avec d'autres leur connaissance de la valeur de l'optométrie.

Par exemple, bon nombre d'entre eux ont des liens politiques plus forts et plus efficaces que la plupart des membres de notre Comité des personnes-clés de l'optométrie. Bon nombre ont de meilleures relations avec le système d'éducation, avec d'autres professions liées aux soins de santé, etc. Dans leur entourage, ils pourront tous transmettre notre message.

Le potentiel d'un groupe de ce type est considérable. L'organisation de ce groupe dans chaque province représentera un défi. Je vous encourage à en parler à votre famille, à votre personnel, à vos patients et à vos amis. Dressez une liste des messagers potentiels de l'optométrie que vous connaissez. Puis convoquez une réunion d'organisation (peut-être, mais sans toutefois que cela soit nécessaire, conjointement avec la prochaine assemblée générale provinciale de votre association).

En fait, je m'attends de recevoir des questions à ce sujet le dimanche 6 mars, à l'occasion de la **Journée de Ligne directe au Président**. Appelez-moi pour en discuter plus à fond. Puis rassemblez tous les autres promoteurs potentiels et mettez-vous en marche!

Votre avenir peut en dépendre.

Le Président,
Scott D. Brisbin, O.D., F.A.A.O.


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