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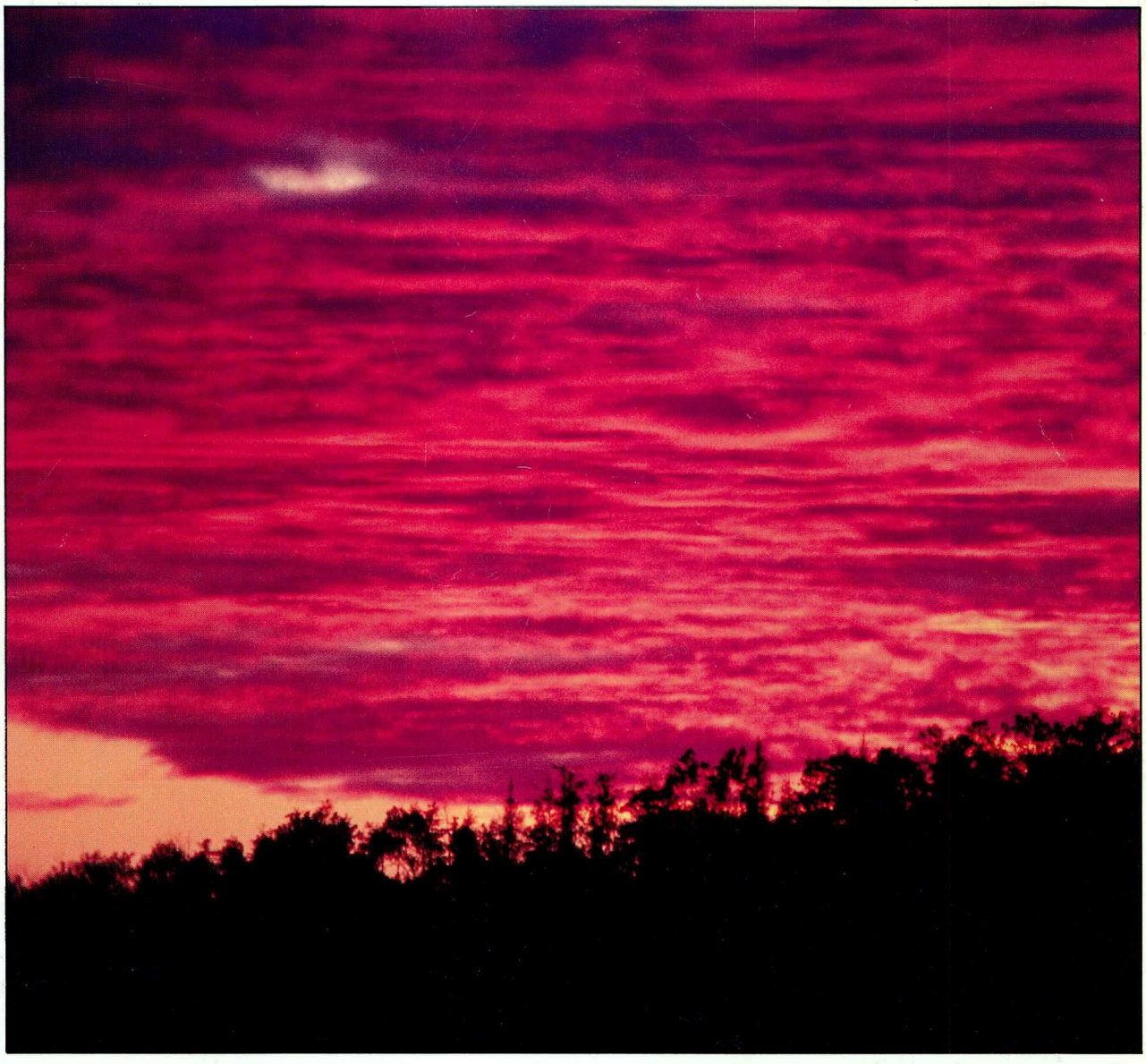

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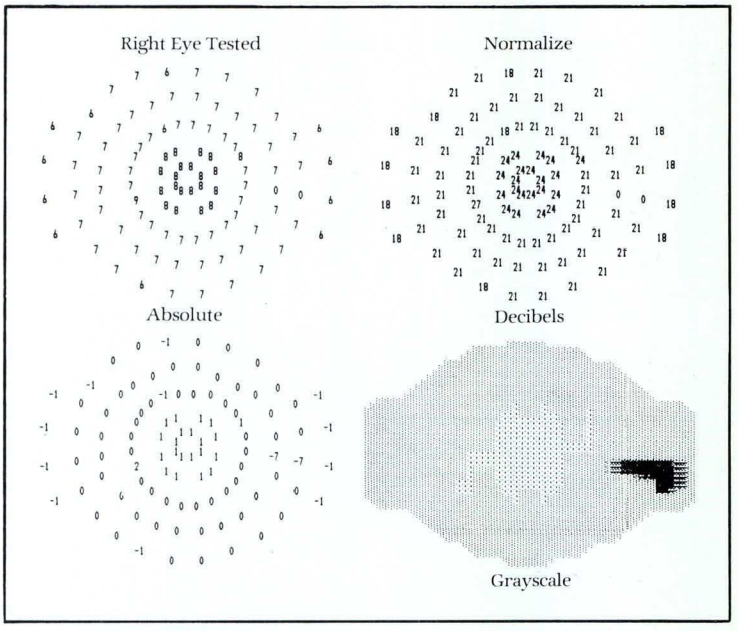

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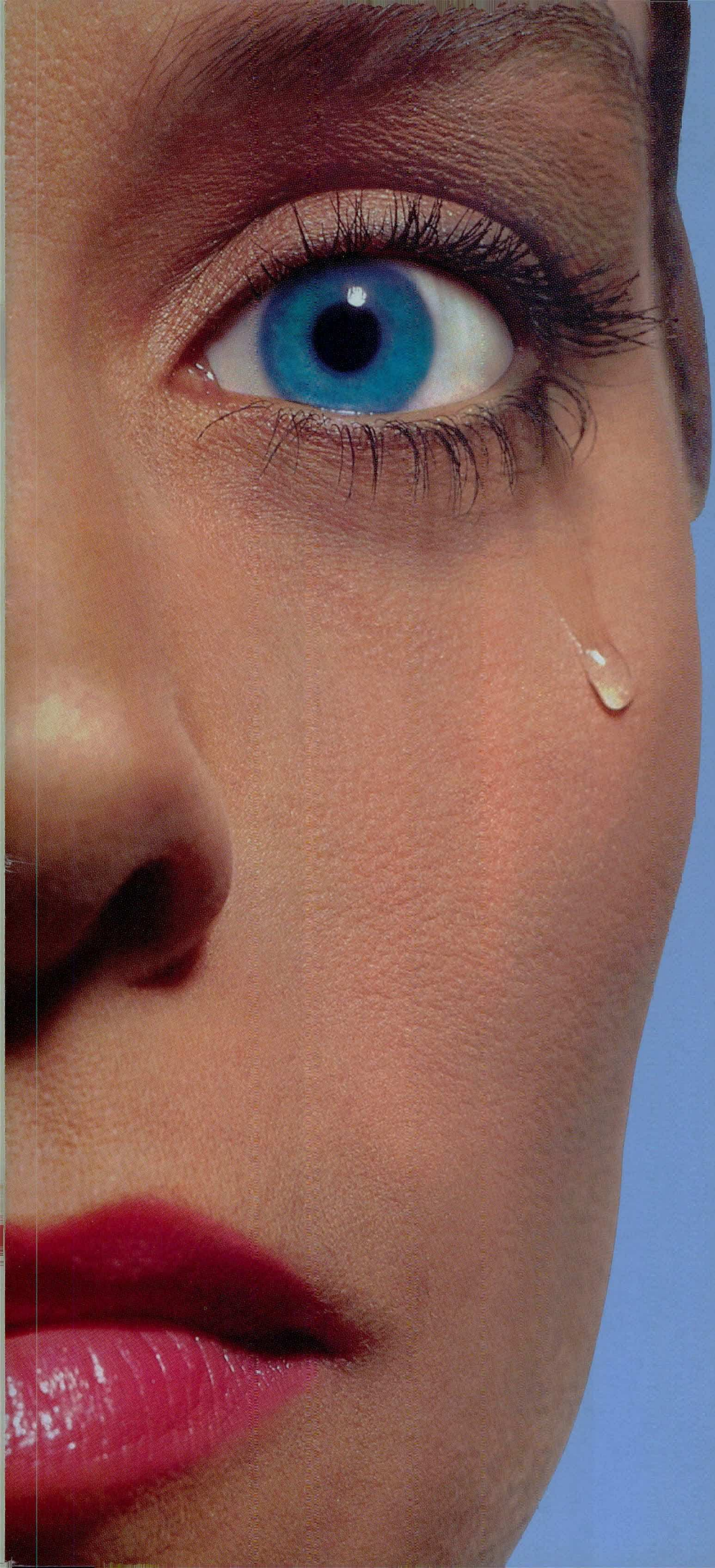

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PRESIDENT'S PODIUM/MOT DU PRÉSIDENT

Your President's Plate is Prodigiously Piled!

"So many issues to comment on — so few issues to comment in!" As I set about jotting down thoughts for the President's Podium in the CJO's Spring Issue that thought, however poor a pun, forced its way onto the paper in front of me. The more I tried to isolate a single theme, the more the other topics on the list screamed for attention. Many simply couldn't wait for the Summer or Fall issue — they'd be stale news by then. Hence, a different format for this PP — a series of thoughts on a number of subjects — a President's Podium Potpourri. (PPP!?)

Our new National headquarters, located on "health care row" in the beautiful Canadian Pharmaceutical Association building in Ottawa, were officially opened April 1st. The opening will be reported elsewhere, but I will say it was a huge success, thanks largely to the efforts of our entire Ottawa staff and Council organizer, Dr. Margaret Hansen des Groseilliers. The success of the move has already become very evident in the months since we left 77 Metcalfe Street. Not only has the move increased the organization of the office and made our Ottawa team a more effective and efficient one, but there has been a positive effect on the image that others have of our profession when they see our new face in the National Capital. (Drop in and see it for yourself when you are next in Ottawa. Your pride in your profession will get a boost. I know mine did.)

Speaking of professional pride, a lot of what we have to be proud of is being celebrated in May at the University of Waterloo. It has been 20 years since the School of Optometry moved from the old College on St. George Street in Toronto to the University of Waterloo. A key participant in creating that milestone in the development of our profession in this country was Dr. E.J. (Ted) Fisher who will be honoured at this year's celebration and convocation when he is invested with the title of Professor Emeritus. I know optometrists from across the country join me in applauding this well-deserved honour and in saying "Congratulations and thank you!" to one of our profession's builders. We have come a long way very quickly with the guidance of leaders such as Ted Fisher and the pace of our accomplishments is ever accelerating. If you can't make it to Waterloo on May 29th, take a minute that day to pause and toast not only a great optometric leader who taught so many of us but also the University which is today the Alma Mater for the majority of optometrists in Canada.

In order to keep pace with the progressive changes facing our profession in "interesting times", a periodic self-assessment is often necessary, albeit sometimes a hazardous exercise. CAO is in the process of just such a reassessment. Reaching right down to our fundamental structure, we are examining and evaluating our relevance over the next decade and beyond under such basic headings as corporate (provincial association) membership, representation by practitioner population, equitable funding formulae and safeguards for jurisdictional integrity. We began several months ago with a solicitation of thoughts from a select group of past and present optometric leaders with a history of involvement with CAO. The provincial association/corporate members will participate in a frank and open exhange of ideas at this year's Congress in Saint John. General members will also have an opportunity for input at the General Business Meeting. Having collected all this input, CAO will then proceed to determine how best to meet the challenges of the next phase of our professional development. Some have experienced concern over meddling with something that "ain't broke". I am convinced, however, that such exercises are vital not only to our professions, but also to each of us as individuals at various points in our lives, if we are to avoid stagnation. The task won't be easy but I, for one, am looking forward to it.

Input to and about CAO comes from many sources via many different vehicles. One of the more interesting ones for me this year, aside from the face-to-face dialogue with many of you I have met during attendance at your Provincial AGMs, came from our first attempt at a "Dial the President Day". Optometrists from coast to coast called me to report on or ask questions about a wide range of optometric topics. I took the opportunity to ask questions too and the feedback I received was most valuable. If "Dial the President Day" had a problem this year, it was our inability to help you remember to call on the appropriate day. I was still receiving calls (and letters) up to two weeks later from optometrists whose best intentions got waylaid on the day itself. We will try it again next year with a more effective awareness campaign. Suggestions are most welcome.

Final item: . . . The Congress is shaping up to be a truly exciting one. The Local Arrangements Committee under the direction of Dr. Barbara Iftody has been enthusiastically designing a surefire memory maker for all of you who make Saint John your destination for August 4th. Continuing Education, politics and fun are all on the menu (see the Preliminary Program elsewhere in this issue). CAO Congresses have traditionally been family affairs. My two teenagers haven't missed one since they were born and they now have friends from coast to coast with whom they correspond, phone periodically and look forward to seeing again at each Congress. It's a great way for youngsters to experience our country with all of its common bonds and its diversities. Take it from someone with a lot of Congresses behind him. Each one is a unique memory you and your family will treasure. Calli and I are looking forward to seeing you in Saint John.

Scott D. Brisbin, O.D.

Votre Président en a plein les bras!

"Tant de questions à simplement commenter et si peu sur lesquelles apporter notre point de vue!" Je commençais à aligner quelques idées pour la Tribune du Président, dans le numéro du printemps de la RCO, mais cette boutade, au demeurant pas très drôle, a commencé à s'imposer : plus j'essayais d'isoler un thème, plus les autres sujets de la liste attiraient mon attention. Nombre d'entre eux ne pouvaient tout simplement pas attendre le numéro de l'été ou de l'automne : ce ne serait plus des nouvelles fraîches. Voici donc une tribune un peu différente, des réflexions sur un certain nombre de sujets, une sorte de "potpourri" de la Tribune du Président.

Le 1er avril, nous avons inauguré officiellement notre nouveau siège social national, sur "l'avenue de la santé", dans le magnifique immeuble de L'association pharmaceutique canadienne de l'industrie du médicament. Vous trouverez plus de renseignements sur l'événement dans un autre article de ce numéro, mais je peux vous dire que ce fut un succès énorme, en grande partie grâce aux efforts de notre personnel d'Ottawa et de l'organisatrice du Conseil, le Dr Margaret Hansen des Groseilliers. Les avantages du déménagement sont déjà très évidents, depuis que nous avons quitté le 77 de la rue Metcalfe. Le bureau est mieux organisé et notre équipe d'Ottawa peut travailler plus efficacement. En plus, notre "nouveau visage" dans la Capitale nationale a eu un effet positif sur l'image que les autres ont de notre profession. (Venez le constater par vous-même, à votre prochain passage à Ottawa. Vous serez encore plus fier de votre profession. C'est en tout cas ce que j'ai ressenti.)

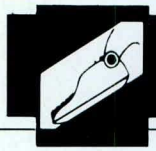
À propos de fierté professionnelle, nous ne pouvons que nous réjouir de la fête qu'on nous prépare, en mai, à l'Université de Waterloo. Cela fait 20 ans que l'École d'optométrie a déménagé de l'ancien collège de la rue St. George, de Toronto, à l'Université de Waterloo. Un des principaux artisans de cette étape historique dans le développement de notre profession au Canada fut le Dr E.J. (Ted) Fisher, qui sera honoré lors des fêtes et de la convocation de cette année : on lui décernera le titre de professeur émérite. Je sais que tous les optométristes du pays se joindront à moi pour applaudir à cet honneur bien mérité et adresser des félicitations et des remerciements à l'un des bâtisseurs de notre profession. Nous avons parcouru rapidement un long chemin, sous la conduite de meneurs comme Ted Fisher et le rythme de nos réalisations est en constante accélération. Si vous ne pouvez être à Waterloo le 29 mai, faites une pause de quelques minutes, cette journée-là, pour porter un toast à un meneur, à un grand optométriste qui a enseigné à un grand nombre d'entre nous, ainsi qu'à l'université qui est aujourd'hui l'Alma Mater de la majorité des optométristes du Canada.

Nous vivons une époque intéressante. Notre profession change progressivement. Afin de rester de son temps, il faut souvent mener une auto-évaluation périodique, exercice nécessaire mais parfois risqué. L'ACO a entrepris une réévaluation de ce genre. En partant de notre structure fondamentale, nous examinons et évaluons notre pertinence, au cours de la prochaine décennie et au-delà, par rapport à des thèmes aussi fondamentaux que les membres collectifs (associations provinciales), la représentation par les praticiens, les formules adéquates de financement et les précautions relatives à l'intégrité des compétences légales. Il y a plusieurs semaines, nous avons, comme première étape, sollicité les réflexions d'un groupe restreint de meneurs passés et actuels de l'optométrie qui se sont occupés de l'ACO. Les membres collectifs et associations provinciales prendront part à un échange d'idées franc et ouvert au congrès de cette année, à Saint-Jean. Les membres ordinaires auront également la possibilité de se faire entendre à la séance d'affaires courantes. Après avoir recueilli tous ces renseignements, l'ACO établira la stratégie répondant le mieux aux défis de la prochaine phase de notre développement professionnel. Certains ont exprimé des réserves, en disant que c'était là se préoccuper de quelque chose qui "n'est pas encore arrivé". Cependant, je suis certain que ce genre d'exercice est essentiel, non seulement pour notre profession, mais également pour chacun d'entre nous, à divers points de notre vie, si nous voulons éviter la stagnation. La tâche ne sera pas facile, mais je ne suis pas de ceux qui la craignent.

L'information qui touche l'ACO ou qui lui est destinée vient de nombreuses sources et par des moyens non moins nombreux. Le moyen que j'ai trouvé le plus intéressant cette année, mis à part le dialogue personnel que j'ai pu entretenir avec nombre d'entre vous au cours des assemblées générales annuelles des associations provinciales, fut notre première journée "le Président vous écoute". Des optométristes de toutes les régions du pays m'ont téléphoné pour discuter avec moi ou me demander mon avis sur une vaste gamme de questions touchant l'optométrie. Évidemment, j'en ai profité pour poser moi aussi des questions et j'en ai tiré des renseignements de grande valeur. Si la journée "le Président vous écoute" s'est moins bien déroulée cette année, cela était dû au fait que nous n'avons pu trouver le moyen de vous rappeler à quelle date téléphoner. Deux semaines plus tard, je recevais encore des appels et des lettres d'optométristes qui, avec les meilleures intentions, s'étaient trompés de jour. Nous recommencerons l'expérience l'an prochain, avec une meilleure campagne de sensibilisation. Vos suggestions sont les bienvenues.

Un dernier point : le Congrès s'annonce très intéressant. Le Comité d'organisation locale présidé par le Dr Barbara Iftody travaille fiévreusement à préparer quelque chose d'inoubliable pour tous ceux d'entre vous qui seront à Saint-Jean le 4 août. L'éducation permanente, la politique et le plaisir sont au menu (voir le programme préliminaire, dans le présent numéro). Traditionnellement, les congrès de l'ACO sont une affaire de famille. Mes deux adolescents n'en ont pas manqué un seul depuis leur naissance. Ils ont maintenant, d'un bout à l'autre du pays, des amis avec qui ils correspondent par téléphone ou par lettre et qu'ils espèrent revoir à chaque congrès. C'est, pour nos jeunes, une façon merveilleuse de connaître notre pays, avec toutes ses diversités et tous ses liens. Croyez-en quelqu'un qui a beaucoup de congrès à son actif : chacun d'eux est un souvenir unique que vous et votre famille conserverez à jamais. Calli et moi avons hâte de vous voir, à Saint-Jean.

Scott D. Brisbin, O.D.



EDITORIAL/ÉDITORIAL

University of Waterloo Honours Pre-Waterloo Graduates

In this year of special recognition by the University of Waterloo to Optometry for its two decades of involvement with the University, the Board of Governors have voted to extend a significant honour to the optometrists who graduated from the Ontario College of Optometry before it made the move to Waterloo in 1967.

A University of Waterloo O.D. degree is being awarded "ad eundem gradum" (literally, "to this level") to those pre-Waterloo OCO graduates who accept the specially extended invitation. The degrees are being presented in conjunction with the 1987 convocation ceremonies which will also see the Waterloo School's first Director, Dr. E.J. Fisher, presented with a singular honour — the designation of Professor Emeritus (as was reported in the CJO • RCO Autumn, 1986 issue).

Marking this unique event in the University's history, Dr. Douglas Wright, President of the University of Waterloo, recently penned a letter to CAO Dr. Scott Brisbin with a personal observation on the granting of the special degrees.

Following is the text of that letter:

I am very pleased to have been asked to make a comment on the University's agreement to grant the honorary degree of *ad eundem gradum* to optometrists. This is the first time that the University of Waterloo has undertaken such a step, but Waterloo has a marvelous record of doing things first and best.

Were it not for the co-operative programs that this University undertook in its first step to provide a different and challenging innovation in post-secondary education, the pathway to exciting and new actions would not be so clear.

And were it not for an event of 20 years ago, this University would not have the School of Optometry as one of its major academic thrusts. I believe it was for that reason, and for others which have to do with our spirit of adventure and innovation, that persuaded the Senate of the University of Waterloo to agree that the honorary degree of *ad eundem gradum* should be made available to those whose educational achievements took place at another time and at another venue.

I am sure that the School and all optometrists and the University will benefit greatly from the continuing association that has been in place now for two decades. The degree *ad eundem gradum* is simply the forerunner of our future.

Douglas Wright
President
University of Waterloo

Twenty Memorable Years

On July 1, 1967, Canada celebrated its centennial birthday party. It was a great nation-wide party with marching bands, parading soldiers, floats, speeches by political leaders, fireworks and celebrations everywhere. Thousands assembled on the lawn of the Parliament Buildings in Ottawa as well as in countless cities, towns and hamlets from coast to coast. Ambitious centennial projects in scores of places were either opened officially or inaugurated. Many parks, sports arenas, library buildings, and community halls were designated as Centennial projects. The great "Expo '67" was in full swing in Montreal. Yes, it was a grand celebration of our country's 100th birthday.

But on that same day, Canadian optometrists were also celebrating at the biennial Congress of the Canadian Association of Optometrists in Montreal. For it had just been announced that, on that day, the former College of Optometry had ceased operation and had become the School of Optometry in the Faculty of Science at the University of Waterloo. After several months of research and negotiation, an agreement had been reached between the Department of University Affairs of the Ontario government, the University of Waterloo, and the College of Optometrists of the province of Ontario. The announcement at the Congress was eagerly received. And so a new era had begun for Optometrical education in Canada.

The months of July and August in 1967 were busy months for the four teaching staff, one secretary and one technician who were charged with the task of moving the College and establishing the new facility. Each person was assigned one area as his responsibility. Students in the course had to be notified to

report to Waterloo in September. Useable equipment, library books, files and records all had to be prepared for moving. Furniture and supplies deemed to be unsuitable had to be put up for sale.

If you have ever moved your home, you will have some idea of the decisions which needed to be made, and the work which had to be done just to vacate the premises at 138 and 140 St. George Street. In Waterloo, space had to be found for clinic, offices and laboratories on a campus already stretched by burgeoning expansion. New equipment and furniture had to be ordered and arrangements made that all would arrive as alterations were completed. Apart from these considerations, faculty members and staff needed to make personal arrangements for moving.

It was a hectic time which will long be remembered by those who were involved. But it was accomplished, and the School was ready to open in early September. (Well, not quite, since there were still construction people in the clinic, and office space was limited, and labs were crowded, but the staff managed.)

And what has happened in the 20 years since that time? From the physical viewpoint, Optometry has a splendid building which is recognized as one of the finest on the campus and, indeed, among North American Optometry buildings. It is furnished with excellent equipment which has been replaced, expanded and updated on a regular basis over the years. The School has developed many new specific areas in patient care: an electro-diagnostic clinic, low vision clinic, binocular vision clinic, pediatric clinic, geriatric clinic, aniseikonia clinic and contact lens clinic, in addition to a high quality primary care clinic. The clinic has a patient record of more than 26,000 visits each year.

An outreach clinic has also been operated, including service to senior citizen's homes and child care facilities in the immediate area. Clinical service has been provided to outlying areas of Canada, involving native Canadians, school and geriatric populations, not only in Ontario, but also in other provinces. Clinical teams have been sent several times to under-developed countries in the Caribbean and in Central America. The University of Benin in Nigeria has received considerable help in establishing the first Optometry program in black Africa, and indeed the only one outside South Africa.

A rapidly growing research program has been developed in basic science research as well as clinical and epidemiological areas. Grants have been received from many private sources and governmental agencies. It would be difficult to name all of the areas involved, but some samples are electrophysiology, sensory perception, colour vision, ainseikonia, border enchancement, geometrical optics, pharmacology, ultra violet radiation, contact lenses, and low vision. Research grants received last year totalled more than $850,000.00. Several hundred papers have been produced at the current rate of 60 per year reporting the results of research. These have been printed in many different refereed journals. This is truly an excellent record among Optometry Schools and Colleges.

The reading room facilities have been expanded with the addition of thousands of new books and a large journal collection. Computers have been installed for student use, as well as a slide viewer, photocopier, and other aids. The reading room study carrels are fully occupied by students literally from dawn to dark. Overflow study tables have been provided in the corridors. A highly efficient library staff is on duty 12 hours a day, with some time on Saturdays and Sundays.

And what of the faculty? There were only 4 full-time teaching faculty in Toronto. Today there are 18, and 8 full time clinical faculty. There are 8 adjunct part-time teaching faculty and 37 part-time clinical faculty members. Through the years, a number of changes have been made in all categories. A few have retired. Some have moved to other institutions — a normal event in Universities today. The support staff numbers some 36 people.

This is a considerable change over the 20 years, and represents a major task just to keep track of the details. Several prominent optometrists, optometrical educators and visual scientists have spent a few terms as visiting professors, while others have visited for a few hours or days.

There have been four directors of the School since it came to Waterloo and each has made a difference but distinct contribution to its growth. At present, the director is also known as the Associate Dean of Science for Optometry. Continued growth is evident in the fact that space is at a premium and several changes have been made from the original plan of the building to accommodate office and research laboratory space.

Finally, the curriculum in Optometry has been under constant study with frequent revisions. It represents all that is best in modern optometrical education. Since coming to Waterloo well over 1000 optometrists have walked across the platform at convocation to receive their "Doctor of Optometry" degrees from the Chancellor and Vice-Chancellor. The majority of these are now serving the Canadian people with superior vision care. They represent more than half of the optometrists in Canada today.

In terms of graduate education, two avenues have been adopted. One involved the establishment of a graduate program in Physiological Optics, leading to the M.Sc. and Ph.D. degrees. This required more than simply drawing up a curriculum and declaring the program ready to accept graduate students. It was first necessary to obtain academic approval within the University of Waterloo itself and, after this, to have the program approved by the Ontario Council on University Affairs. The process was completed in two stages and each involved more than two years of work and negotiation. The second method of providing graduate education was to establish advanced clinical training for those who wished to become clinical teachers, or who wished to acquire special skills in some particular area of Optometry. This was done by establishing clinical residencies. Approximately a dozen graduate degrees have been awarded, and more than twenty graduates have completed the residency program.

With a worthy heritage, an excellent faculty and a fine, but crowded, facility, the future of Optometrical education at the University of Waterloo is very promising indeed. The profession can well be proud of the present and past faculty and staff of the School who have served and are still serving as a team dedicated to developing the program.

Edward J. Fisher, M.A., D.Sc., F.A.A.O.

From St. George Street to Columbia Avenue – Twenty Years Already!

"There is a tide in the affairs of men which, taken at the flood, lead on to fortune."

How true and applicable are the old bard's words in the story of Optometry. Foresight, organization, education and hard work are the stepping stones upon which Optometry has built its good fortunes.

These stepping stones did not just happen on our way. They came to be there because optometric pioneers and leaders sought to improve the quality of their services and, over many years, strove diligently and unselfishly to build the "House of Optometry".

> "There is a tide in the affairs of men which, taken at the flood, lead on to fortune."

Historians will recall the founding of the Ontario Association in 1909, the Justice Hodgins report on medical education in Ontario in 1917, which led in turn to the first Optometry Act in Ontario in 1919, the institution of the first Optometry program at the Central Technical High School in 1921 and the founding of the College of Optometry of Ontario in 1925.

We can recount the evolution of our training from a two-year course (1925 - 1936), to a three-year program (1937 - 1956), to a four-year curriculum yielding a doctorate degree and the gradual fleshing out of the program to include those subjects which made Optometry a true primary care discipline.

> And so the stepping stones have become the building blocks and our "house" stands firm. But four walls do not in themselves make a home. It is what goes on inside those walls that converts a mere building into a home.

We cannot omit to mention our concerns about the need for optometric research in vision care and our aspirations to attain university status and full academic recognition.

And so the stepping stones have become the building blocks and our "house" stands firm. But four walls do not in themselves make a home. It is what goes on inside those walls that converts a mere building into a home.

Twenty years have passed since the doors at 140 St. George were closed for the last time. We did not fold our tents and silently steal away but went rather with joy in our hearts, a spring in our step and great expectations. We moved to new horizons well aware that continued progress would be just as demanding, even more demanding of our efforts than in the past.

It is with enthusiasm and joy that we celebrate the 20th Anniversary of the School of Optometry, Faculty of Science, University of Waterloo. We take pride, too, in the preceding Guest Editorial, recounting those happenings which turned a building into a new and permanent home for Optometry, by a "raconteur" who had so much to do in making these "happenings" happen, and who so aptly deserves the title "Professor Emeritus".

> It is with enthusiasm and joy that we celebrate the 20th Anniversary of the School of Optometry, Faculty of Science, University of Waterloo.

To Professor E.J. Fisher and his close collaborators, C.W. Bobier, W.S. Long, W.M. Lyle and M.E. Woodruff, to those many other faculty and staff whose dedication created our home, to the authorities at the University of Waterloo, particularly former President W.G. Hagey and Dean W.A.E. McBride, who accepted Optometry as one of their own and provided the moral support and atmosphere so essential to a happy home life,

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20th Biennial Congress
August 4 - 7, 1987

20e congrès biennal
4 - 7 août 1987

WELCOME!

Dear Colleague

It is with great pleasure and anticipation that I invite you on behalf of the New Brunswick Association of Optometrists to join with us in Saint John, August 4-7, 1987, as we host for the first time the Biennial Congress of the Canadian Association of Optometrists. This is the twentieth Biennial Congress, one which we hope to fill with lots of "Down East" hospitality and bilingual atmosphere.

The local arrangements committee is chaired by Dr. Barbara Iftody. I am sure that she and all her committee members will feel their efforts amply rewarded if you and your family attend and enjoy all the Congress has to offer. We want to particularly welcome all companions and children to this special time, our "Merry-Tyme Mingle".

You will find included in this letter a registration form and a sketch of Congress events for all. Please note the early registration dates, and let us know that we will be able to be your hosts in all events from a "lobster feed", to the C.A.O. sections meeting, and the Continuing Education sessions. A duplicate registration form and further details are to be found in the Canadian Journal of Optometry.

Sincerely yours

Dr. L.F. Clements
President, N.B.A.O.

BIENVENUE!

Chèr(e) collègue,

J'ai le plaisir et l'honneur de vous inviter, au nom de l'Association des optométristes du Nouveau-Brunswick, à venir vous joindre à nous à Saint John du 4 au 7 août 1987 puisque nous sommes, pour la première fois, l'hôte du congrès biennal de l'Association canadienne des optométristes. Il s'agit de la vingtième biennale, un congrès qui, espérons-nous, vous fera vivre l'hospitalité de l'"Est" dans une ambiance bilingue.

Le Comité d'organisation locale est présidé par le Dr Barbara Iftody. Je suis persuadé qu'elle même et tous les membres de son Comité seront très heureux de vous accueillir avec votre famille et de vous faire profiter de tous les services spéciaux que le congrès vous réserve. Nous voulons souhaiter une bienvenue particulière à tous les conjoints et à tous les enfants et nous les invitons à "venir voir comme on s'amuse".

Vous trouverez ci-inclus une formule d'inscription et un programme sommaire des activités du congrès pour tous. Veuillez noter les dates de pré-inscription et nous indiquer si nous aurons le plaisir de vous accueillir à toutes les activités qui comprennent aussi bien un "souper au homard" que la réunion des sections de l'ACO et les ateliers de formation permanente. Vous trouverez également une autre formule d'inscription ainsi que d'autres détails dans la Revue canadienne d'optométrie.

Je vous prie d'agréer l'expression de mes sentiments distingués.

Dr L.F. Clements
Président, A.O.N.B.

Congress at a Glance

Optometrists and Spouses/Guests — registration starts at noon on Tuesday, Aug. 4, with opening ceremonies from 7 to 10 pm. A lobster feast and fun night follows on Wednesday, Aug. 5 from 6 pm. to 1 am. Thursday night has been set aside for class reunions. After the Friday noon C.O.E.T.F. reception which is for both O.D.'s and spouses/guests, the afternoon is left as free time.

Spouses/Guests — A few featured events, in addition to those held jointly with the optometrists include a day trip to scenic St. Andrews by the Sea with a lunch at the Algonquin Hotel and Saint John bus and walking tours. The hospitality room will be open as a meeting place, with coffee and information. Friday morning will include time at the Saint John Aquatic Centre and a visit with Pete of Pete's Frootique guaranteed to be informative and lively.

Junior Delegates 1 — will be the pre-schooler group from infants to five years. These children will have programmed daycare activities in one of the convention hotels all day, each day of the convention. Please inform us on the registration form if babysitters will be needed for the evenings.

Junior Delegates 2 — will be the age group 6-11 years. Some events include a pyjama party, bowling, day trip to St. Andrews, waterslides, swimming and a trip to the zoo!

Junior Delegates 3 — will be for ages 12 and up. They will have a two-day overnight stay in St. Andrews. This will be hosted by Sunbury Shores Arts and Nature Centre. They will join with the junior 2 group for some events such as swimming and waterslides but will have their own farewell party on Friday night.

Coup d'oeil sur le congrès

Optométristes et conjoint(e)s — L'inscription commence à midi le mardi 4 août, les cérémonies d'ouverture devant avoir lieu de 19 h00 à 22 h 00. Le tout sera suivi d'un souper au homard et d'une soirée mondaine le mercredi 5 août de 18 h 00 à 1 h 00 du matin. La soirée du jeudi est réservée aux réunions de classe. Après la réception du FFOCE, qui se tiendra vendredi à laquelle sont conviés les optométristes et les conjoints, l'après-midi sera libre.

Les conjoint(e)s — Les activités prévues en plus de celles qui auront lieu simultanément avec les optométristes comprennent une visite d'une journée à St. Andrews-sur-mer y compris le déjeuner à l'Hôtel Algonquin ainsi que des visites à pied et en autobus de la ville de Saint John. La suite de réception sera ouverte; elle servira de lieu de rencontre où vous pourrez vous procurer de l'information tout en prenant votre café. Une partie de la matinée du vendredi sera passée au Centre aquatique de Saint John; ensuite, on a prévu une visite guidée par Pete de Pete's Frootique, activité qui devrait être très informative et intéressante.

Jeunes délégués 1 — Le groupe d'âge préscolaire à partir des bébés jusqu'aux enfants âges de cinq ans. Ces enfants pourront participer à des activités de garderie organisées dans l'un des hôtels du congrès pendant toute la journée, tous les jours du congrès. Veuillez nous indiquer sur la formule d'inscription si vous prévoyez avoir besoin de services de gardiennes le soir.

Jeunes délégués 2 — Les enfants âges de 6 à 11 ans. Plusieurs activités sont prévues pour ce groupe, y compris une partie pyjama, des parties de quilles, une visite d'une journée à St. Andrews, des glissades d'eau, de la natation et une visite au zoo.

Jeunes délégués 3 — Les enfants âgés de 12 ans et plus. Ils feront une visite de deux jours à St. Andrews. Cette visite est parrainée par le Sunbury Shores Arts and Nature Centre. Ils se joindront au groupe des jeunes 2 pour certaines activités telles que la natation et les glissades d'eau, mais ils auront leur propre soirée d'adieu le vendredi soir.

Education/Business Program

Tuesday, August 4	
7:00 pm to 10:00 pm	Opening Ceremonies
Wednesday, August 5	
9:00 am to 12 noon	Education Session
12 noon to 2:00 pm	Welcome Luncheon
2:00 pm to 5:00 pm	Education Session
Thursday, August 6	
9:00 am to 12 noon	Education Session
12 noon to 2:00 pm	Exhibitors Luncheon
2:00 pm to 5:00 pm	C.A.O. Sections Meeting
6:00 pm to 8:00 pm	Exhibitors Cocktail Period
8:00 pm to ?	Class Reunions
Friday, August 7	
9:00 am to 12 noon	C.A.O. General Business Meeting C.O.E.T.F. Launching of Phases II & III
6:30 pm to 12 midnight	C.A.O. President's Reception, Banquet Awards and Ball

Programme d'éducation et d'affaires

Mardi 4 août	
19h00-22h00	Cérémonies d'ouverture
Merdredi 5 août	
9h00 - 12h00	Séance de formation
12h00 - 14h00	Déjéuner d'acceuil
14h00 - 17h00	Séance de formations
Jeudi 6 août	
9h00 - 12h00	Séance de formation
12h00 - 14h00	Déjeuner des exposants
14h00 - 17h00	Réunions des sections de l'A.C.O.
18h00 - 20h00	Cocktail pour les exposants
20h00 - ?	Réunions de classe
Vendredi 7 août	
9h00 - 12h00	Assemblée générale de l'A.C.O. Lancement des phases II & III du F.F.O.C.E.
18h30 - 24h00	Cocktails, réception, banquet et danse du président de l'A.C.O. et prix

FEDERAL ISSUES/QUESTIONS FÉDÉRALES

CAO . . . Unveiling Our New Face And Our New Place

CAO Councillors at the opening pose reflectively in the Boardroom. Front row, L-R: Dr. Tom Adamack, Dr. Scott Brisbin, Dr. Bruce Rosner. Back row, L-R: Dr. Mike Duffey, Dr. Margaret Hansen des Groseilliers, Dr. Jim Krueger, Dr. Grant Campbell, Dr. Doug Côté, Dr. Greg Perkins.

Although actually open for business on November 23, 1986, CAO's new Ottawa offices were finally opened **officially** with a special reception and ceremony on April 1, 1987. Guest of Honour, Deputy Minister of National Health & Welfare, Dr. Maureen Law attended and complemented this welcome opportunity to greet our many new neighbours on Ottawa's "Health Care Row" and beyond.

The opening was held in conjunction with the Spring, 1987 meeting of the Council of the Canadian Association of Optometrists and Councillors, sporting white carnation boutonnieres, hosted over 100 guests. The attendees included representatives of most of the major players on the Federal Health Care scene, among them, the Canadian Medical Association, the Canadian Dental Association, the Canadian Nurses' Association, the Canadian Red Cross, the Canadian Chiropractic Association, and many, many others.

Also being held concurrently with the national Council meeting was the Ontario Association of Optometrists' Annual Congress, held this year in Ottawa. As a result, a strong optometric presence was ensured and the profession was represented not only by invited members of the Ottawa and Outaouais Societies of Optometrists, but also by l'Association des Optométristes du Québec and the OAO's entire Board of Directors.

In addition to CAO Councillors, the CAO national office staff acted as hosts and, as guests arrived in the foyer of 1785 Alta Vista Drive, they were given name badges and directed to the third floor by Annette McDonald and Deanna Verhey. Then, as they left the elevator, they were welcomed, musically, by gentle tones provided by a professional harpist and, more directly, by two other CAO staff members, Chantal Wall and Ruth Wilcox.

Informality was the measure of the evening and the first hour was spent sampling a superb hors d'oeuvres buffet arranged by Dave Smith, one of the capital's foremost caterers and Ottawa's Small Businessman of the Year for 1987.

As a memento of her participation, Dr. Maureen Law was presented with a framed copy of the 1987 Save Your Vision Week poster by President Dr. Scott Brisbin.

CAO President Dr. Scott Brisbin's welcoming remarks to the assembled guests keynoted the Association's anticipation of many future productive years of beneficial relationships on behalf of the profession, in the common cause of delivering health care for Canadians.

CAO was especially pleased that an invitation to attend the official opening had been accepted by our Guest of Honour. As Deputy Minister of National Health and Welfare, Dr. Maureen Law's speech provided a special insight into the goals and priorities of her department. Among them were a number of issues involving current optometric researchers as well, such as child abuse, smoking and its effects on health in general and vision in particular and the curtailing of the transmissibility of the AIDS Virus.

Following her remarks, Dr. Law conducted an official "unveiling" of the CAO Logo and Office Sign. Her formal functions concluded, Dr. Law was then able to join in the evening's socializing and discovered very quickly how much common ground exists in the activities of organized Optometry, and those of the federal Ministry of Health. CAO's Executive Director, Gérard Lambert, introduced Dr. Law to Dr. Emerson Woodruff, whose own research into (i) the effects of smoking on pre-natal development of the visual system and incidence of problems after birth and (ii) child abuse and visual problems served to underscore the valuable potential future role for optometric consulting services to the Federal Government.

CAO's recently renewed thrust into the government affairs / political action arena has been long awaited by our corporate members, the ten provincial optometric associations, and by **their** members, the optometrists of Canada.

Since its inception, the Canadian Association of Optometrists has fulfilled many roles in support of the profession's growth to its present status as one of this country's major providers of primary health care.

Our evolution has shaped the national Association into a central body providing a wide variety of membership and administrative services to its confederation of provincial associations. It has, of necessity, been a gradual process. Yet even during these years, CAO, due to the efforts of uncounted (and unsung) dedicated optometrists, has recorded notable successes in the political action field.

Now, as ever stronger provincial associations provide more specifically focussed membership, administrative and political action programs, CAO Council has decided to assess, perhaps more thoroughly than has ever been done before by the Association the role and structure of the national body.

It is expected that one result may well be a mandate to chart a new course,

A unique musical touch was added to the evening by professional harpist Mme. Joanne Meis.

redefining and reinforcing CAO's relationship not only with its own corporate members and Canada's optometrists, but also with the Federal Government and many of the non-governmental groups and individuals with whom we have had contact over the years.

At the root of this massive exercise in self-evaluation is the underlying principle that, as a relatively small membership organization with a tremendously ambitious agenda, in spite of limited resources, the more concentrated our focus, the more effective we become.

That new focus took a giant step forward with CAO's relocating its national headquarters to "Health Care Row" in Ottawa. At the same time, the introduction of a more efficient computerization program has also streamlined our communications and administrative processes.

Meanwhile, literally hundreds of optometrists have been stepping forward to identify their political contacts and join our KEYPERSON team. We are looking forward to enjoying the fruits of these labours in the years ahead, where Optometry and optometric vision and eye care will serve as a prime resource of health care information to every level of government, and be recognized as such.

Achieving such recognition is a primary goal for CAO. The directions travelled in recent months have greatly increased our profile in Ottawa, steps which can only bode well for the profession. Indeed, they are prerequisites for our sustained growth and well being.

B. Alex Saunders
Governmental Affairs Co-ordinator

L'ACO... Un nouveau visage, un nouveau siège social

Bien que notre nouveau siège social d'Ottawa ait commencé à fonctionner le 23 novembre 1986, ce n'est que le 1er avril 1987 qu'il a été **inauguré** officiellement, dans le cadre d'une cérémonie et d'une réception spéciales. L'invitée d'honneur et sous-ministre de la Santé nationale et du Bien-être social, le Dr Maureen Law, était avec nous et a profité de cette excellente occasion pour saluer nos nombreux nouveaux voisins de "l'avenue de la santé d'Ottawa" et des environs.

L'inauguration avait lieu en même temps que la réunion du printemps 1987 du Conseil de l'Association canadienne des optométristes et conseillers : ils étaient plus d'une centaine d'invités, fleur à la boutonnière. L'assistance comptait des représentants de la plupart des grands intervenants de la scène fédérale des soins de santé, notamment l'Association médicale canadienne, l'Association dentaire canadienne, l'Association des infirmières et infirmiers du Canada, la Croix-Rouge du Canada, l'Association chiropratique canadienne, etc.

Tout le personnel du bureau national de l'ACO était présent : (de gauche à droite) Gérard Lambert, Ruth Wilcox, Alex Saunders, Annette McDonald (assise au bureau), Mike DiCola, Chantal Wall et Deanna Verhey.

En même temps que la réunion du Conseil national, avait lieu cette année à Ottawa le Congrès annuel de l'Association des optométristes de l'Ontario (OAO). Ainsi, l'optométrie était fortement représentée, de même que la profession, non seulement par les membres invités des sociétés d'optométristes d'Ottawa et de l'Outaouais, mais également par l'Association des optométristes du Québec et l'ensemble du Conseil de direction de l'OAO.

Les conseillers de l'ACO et le personnel du bureau national ont rempli le rôle d'hôte et, à mesure que les invités arrivaient dans le hall d'entrée du 1785 de la promenade Alta Vista, ils leur remettaient leur macaron d'identification et Annette McDonald et Deanna Verhey les dirigeaient vers le troisième étage. À leur sortie de l'ascenseur, les visiteurs étaient accueillis par la musique délicate d'une harpe (jouée par une artiste professionnelle) et, plus directement, par deux autres membres du personnel de l'ACO, Chantal Wall et Ruth Wilcox.

Le Dr Margaret Hansen des Groseilliers et le Dr Scott Brisbin, souriants et détendus : leurs fonctions officielles de la soirée sont probablement terminées.

La détente fût la marque de cette soirée. Les invités ont passé la première heure à goûter les hors-d'oeuvre d'un magnifique buffet organisé par Dave Smith, l'un des traiteurs les plus réputés de la Capitale et qui a reçu, en 1987, le titre de petit entrepreneur de l'année, à Ottawa.

Le Dr Maureen Law enlève le voile qui cachait l'enseigne et le logotype du nouveau bureau.

Le mot de bienvenue du Dr Scott Brisbin, président de l'ACO, avait pour thème les nombreuses années productives de rapports bénéfiques que compte entretenir l'Association, au nom de la profession et pour la cause commune des soins de santé aux Canadiens.

L'ACO a été particulièrement heureuse que l'invitée d'honneur ait accepté d'assister à l'inauguration officielle. Le discours du Dr Maureen Law, sous-ministre de la Santé nationale et du Bien-être social, a été particulièrement riche de renseignements sur les buts et les priorités du ministère. Elle a traité notamment d'un certain nombre de questions qui ont des incidences sur les recherches actuelles en optométrie, notamment la violence envers les enfants, le tabagisme et ses effets sur la santé en général et surtout sur la vue, ainsi que les recherches visant à empêcher la transmission du virus du SIDA.

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Après son allocution, le Dr Law a "dévoilé" officiellement le logotype et l'enseigne du bureau de l'ACO. Sa mission officielle remplie, le Dr Law s'est jointe aux discussions des personnes présentes et a rapidement découvert que les associations d'optométrie et le ministère fédéral de la Santé avaient énormément de points en commun, sur le plan des activités. Gérard Lambert, directeur général de l'ACO, a présenté le Dr Law au Dr Emerson Woodruff, dont les recherches, sur (i) les effets du tabagisme sur le développement prénatal du système visuel et l'incidence des problèmes après la naissance et (ii) la violence envers les enfants et les problèmes visuels, ont servi à faire ressortir les pos-

Mini-sommet dans le bureau de direction : (de gauche à droite) le Dr Scott Brisbin, le Dr Greg Perkins, le Dr Tom Adamack et le Dr Grant Campbell.

Il a fallu un peu d'encouragement pour décider le Dr Maureen Law à couper le magnifique gâteau fait et décoré spécialement pour marquer l'événement.

sibilités futures des services-conseils en optométrie pour le gouvernement fédéral.

L'orientation que l'ACO vient de se redonner, en matière d'intervention dans le domaine des affaires gouvernementales et de l'action politique, était depuis longtemps attendue par nos membres collectifs, c'est-à-dire les dix associations provinciales d'optométrie, ainsi que par **leurs** membres, les optométristes du Canada.

Depuis sa création, l'Association canadienne des optométristes a assumé de nombreux rôles, pour appuyer la croissance de la profession jusqu'à sa situation actuelle : elle est maintenant l'un des principaux fournisseurs de soins fondamentaux de santé du pays.

Notre évolution a fait de l'Association nationale un organisme central fournissant toute une gamme de services administratifs et professionnels à sa confédération d'associations provinciales. Par nécessité, cela s'est fait graduellement. Pourtant, même pendant ces années, l'ACO, grâce aux efforts d'innombrables optométristes dévoués (et souvent inconnus), a pu enregistrer des succès notables sur la scène politique.

Désormais, les associations provinciales, toujours plus fortes, fournissent des services professionnels, administratifs et d'action politique plus spécifiques et, pour cette raison, le Conseil de l'ACO a décidé de mener une évaluation, peut-être la plus approfondie qu'ait jamais entreprise l'Association, du rôle et de la structure de l'organisme national.

Il en résultera peut-être le mandat de tracer une nouvelle orientation, de redéfinir et de renforcer les rapports de l'ACO, non seulement avec ses propres membres collectifs et les optométristes du Canada, mais également avec le gouvernement fédéral et nombre de groupes non gouvernementaux et de personnes avec lesquels nous avons entretenu des rapports au fil des ans.

À la base de cette auto-évaluation en profondeur se trouve le principe suivant : en tant qu'organisme comptant un nombre relativement faible de membres, mais qui a de vastes projets malgré ses ressources limitées, plus l'ACO concentrera ses efforts, plus elle sera efficace.

Cette nouvelle orientation a fait un grand pas en avant avec le déménagement du siège social national de l'ACO sur "l'avenue de la Santé" d'Ottawa. En même temps, nous avons rationalisé nos modalités de communication et d'administration par l'introduction d'un programme d'informatisation plus efficace.

Dans l'intervalle, littéralement des centaines d'optométristes se sont avancés pour nous faire connaître leurs contacts politiques et se joindre à notre équipe de personnes-ressources. Nous avons hâte de voir les fruits de leurs efforts, dans les années à venir, quand l'optométrie, la vision optométrique et les soins occulaires seront parmi les principales ressources d'information, en matière de soins de santé, et seront reconnus comme tels à tous les paliers de gouvernement.

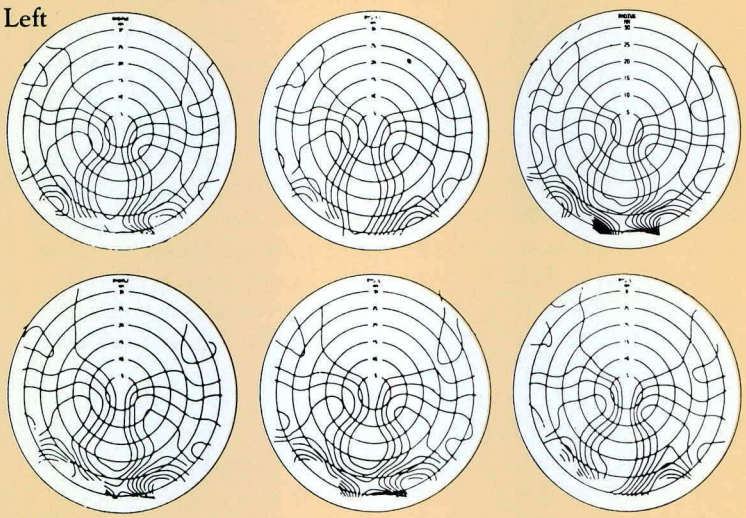
Parvenir à cette reconnaissance est un des grands objectifs de l'ACO. Les orientations prises ces derniers mois ont grandement amélioré notre position à Ottawa et cela ne peut que bien augurer pour la profession. En fait, ce sont des conditions fondamentales, pour le maintien de notre croissance et notre mieux-être.

Le coordonnateur des affaires gouvernementales,
B. Alex Saunders

Une fois le gâteau coupé . . . la dégustation officielle : (de gauche à droite) Gérard Lambert, le Dr Scott Brisbin, Calli Brisbin, le Dr Maureen Law.

Before you order your here's something

Left



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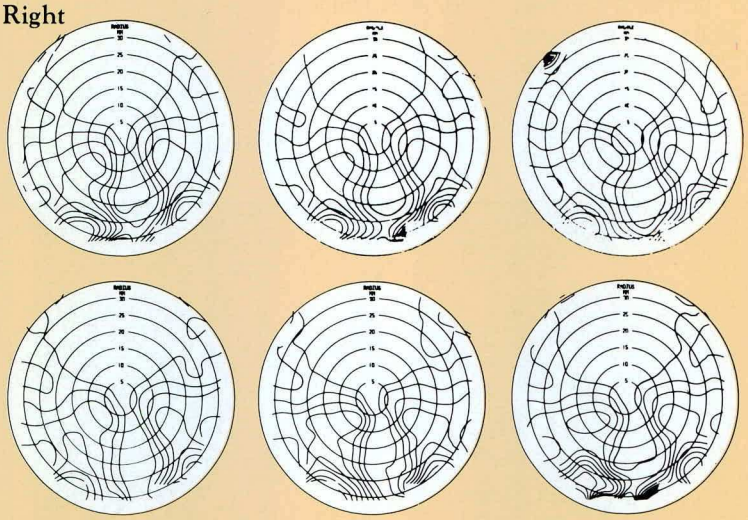


12 "identical" Varilux 2 lenses.

If you've been having trouble working with Varilux 2 lenses, we're not surprised.

Just look to the left and you'll see why.

These are computer-generated contour maps of 12 seemingly "identical"* Varilux 2® lenses.

Measured on a lensmeter, all 12 yield the same result—plano distance, +2 add. But computer plots of their actual surface designs tell quite a different tale.

As you can plainly see, they aren't "identical" at all.

Viewing areas differ in size and in shape, especially in the near and intermediate zones. And the astigmatic patterns vary from lens to lens.

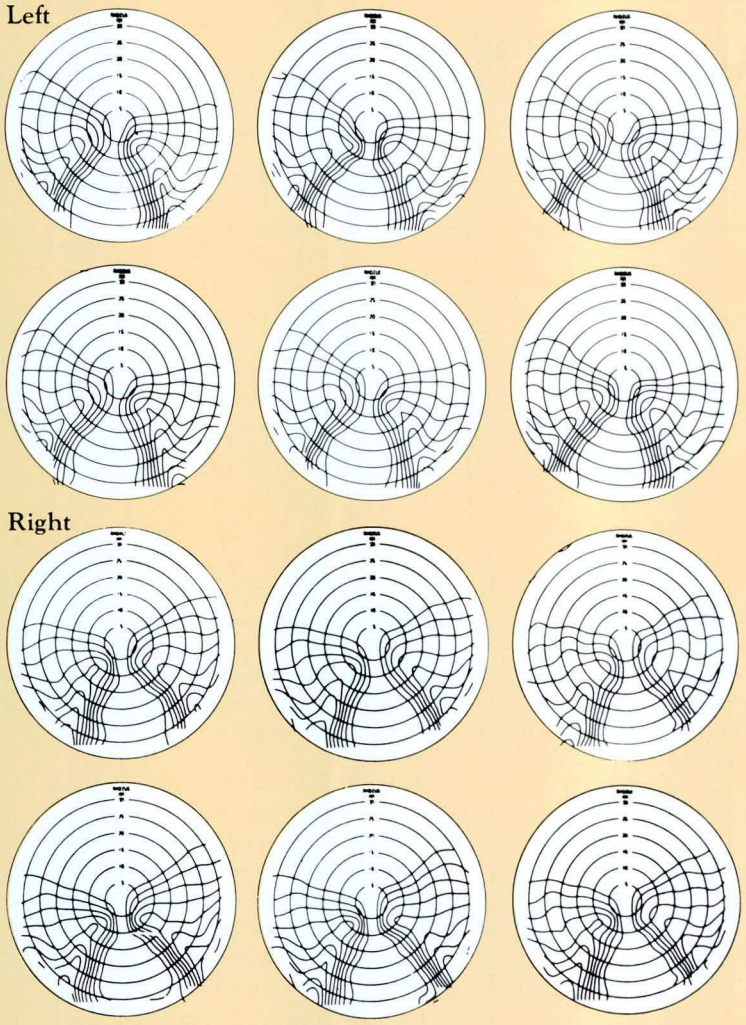
Inconsistencies like these can cause problems in dispensing. If a "pair" of lenses aren't alike you may have trouble with fitting and with adaptation, especially on your more demanding patients.

Maybe you should look for a more reliable progressive, like the one at right . . .

*Twelve Multi-Optics Varilux 2 and twelve Sola VIP lenses were obtained from ordinary shelf stock. The lenses were all surfaced to a plano distance, +2.00D near add. Using a standard measuring device the data was processed by a computer and the computer then plotted the surface astigmatism of each lens so the results could be analyzed.

The first contour line plotted, bordering the distance, intermediate and near vision zones denote astigmatism of about 0.50D or more. Each succeeding contour line marks an increase of 0.50D of astigmatism.

next pair of progressives, you should see.



12 "identical" Sola VIP lenses.

We hate to be blunt but the situation here is a whole lot better.

Here's the progressive you don't have to worry about . . . the consistent and reliable Sola VIP.™

These are 12 "identical"* Sola VIP lenses.

Just like the other brand, their lensmeter results were the same: plano distance, +2 add.

But unlike the other brand, the computer-generated contour maps of VIP lenses *all look alike.*

All the nears are extra-large, and the intermediates leave ample room for easy fitting. The shapes are all the same, too, with gently changing curves to minimize peripheral distortion.

In every lens.

This consistent design allows consistently successful dispensing. Without mis-fittings and endless follow-ups. Without worry or wasted time — for you or your patients.

So before you order your next progressives, remember what you saw here. The lens you can rely on is Sola VIP.

SOLA

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Sola Optical USA, Inc., (800) 358-8258 (Continental U.S.), (800) 862-4934 (California), or (707) 763-9911.

CLINICAL RESEARCH/RECHERCHE CLINIQUE

Nerve Head Anomaly Associated with Pituitary Tumor

T.D. Williams *

**O.D., M.S., Ph.D.*
School of Optometry
University of Waterloo

A 54-year old Caucasian female was seen in the School of Optometry, University of Waterloo clinic for field testing to rule out possible glaucoma. She had had a pituitary tumor (histologically confirmed as a chromophobe adenoma) removed 10 years previously. Field testing and fundus inspection ruled out glaucoma: there were no nasal steps and no scotomas in the arcuate areas.

Field testing (Figs. 1 and 2) did indicate some midline-type superior bitemporal defects. Moreover, the blindspots on field testing were displaced down and out (although the patient has only a modest myopia (around 1.5 D). Ocular fundus examination revealed a fairly striking situs inversus OS[1] (Fig. 3).

Comment:

The visual field findings in this case serve to remind the clinician that targets such as the Goldmann I-2 target (or finer, dimmer targets) should be used when seeking midline-type defects, as the stronger target (Goldmann I-4) failed to show a midline cut in the isopter. The Goldmann I-2 target is reasonably equivalent to a 1/1000 W target at the tangent screen, provided the screen illuminance is on the order of 108 to 160 L/sq m (10 to 15 foot-candles).

Age-matched normal perimeter data are shown with short broken lines in Figs. 1 and 2. The inner pattern of short broken lines is a normal I-2 isopter,

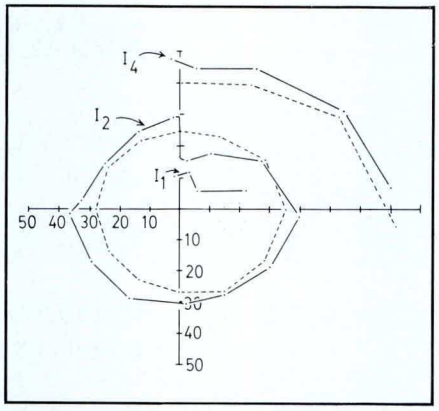

Fig. 1 Results of visual field testing for right eye. Three test stimuli were used: Goldmann I-2 (equivalent to a 1/1000 W target at the tangent screen when illuminance = 10 to 15 foot-candles), Goldmann I-1 (0.5 log units dimmer than I-2) and Goldmann I-4 (1 log unit brighter than I-2). A complete isopter (indicated by arrow) was plotted using the I-2 stimulus. The other two stimuli were used to assess the superior temporal portions of the field (incomplete isopters marked by arrows). For comparison, short dotted lines indicate normal age-matched normal results for the I-2 stimulus (complete normal isopter shown), and for the I-4 stimulus (partial isopter shown). The I-2 isopter shows a definite break at the midline: there is a superior temporal loss here. The dimmer I-1 target revealed not only a superior temporal loss, but an overall depression of the superior field. The brighter I-4 target showed no field abnormality.

while the outer, incomplete pattern of short broken lines is from a normal I-4 isopter. Note that the patient is showing a larger-than-normal isopter in three quadrants out of four: this makes the superior temporal loss even more apparent. Note further that the patient's I-4 data match the normal data fairly closely in the superior temporal quadrants, with no disturbance at the midline.

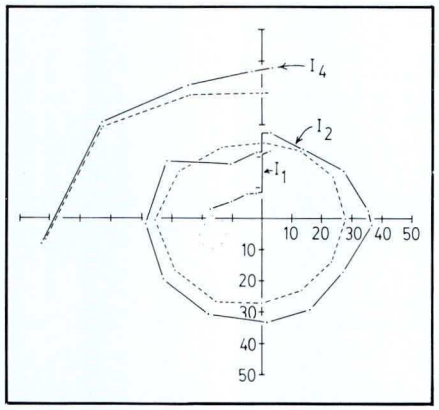

Fig. 2 Similar findings for the left visual field. Here, the I-1 target shows a loss with less of an overall superior character and more of a superior temporal nature.

The I-4 blindspots are shown as a pattern of unconnected dots: they are displaced in an outward and inferior direction. Such a finding is not uncommon in relatively high myopes (say greater than 5D), but the patient has only a modest degree of myopia (1.5 D). This prompts a closer examination of the fundus photos. Ordinarily, a horizontal line passing through the center of the fovea will cut across the nerve head about ⅓ of the way up from the inferior edge. This does not occur in these photos. This suggests a rather inferior positioning of the fovea relative to the nerve head. The right nerve head appears to be smaller than the left.

Taylor[2] reported on an association between tumors affecting the anterior visual pathway and malformations of the optic nerve head. The tumors found in his seven cases included glioma, astrocytoma (one pilocytic and one fibrillary), a suprasellar mass (which was not biopsied), and craniopharyngioma. The

associated malformations of the nerve head included conus (frequently inferior), dysversion of the disk, situs inversus of the central retinal vessels, and hypoplasia of the nerve head (both overall and segmental).

Keane[3] reported a further two cases in which tumors were found in the chiasmal region (one was a chromophobe adenoma, the other a pinealoma): in the first case, one disk appeared somewhat hypoplastic, while the other showed an inferior nasal conus; in the second, the left eye showed a strong superior nasal oblique direction of the optic nerve, with an associated inferior temporal conus. He felt that the nerve head findings were unrelated to the tumors.

This case would appear to be another instance of an association between a malformation of the optic nerve (situs inversus) and a tumor affecting the anterior visual pathway, in this case a pituitary tumor (chromophobe adenoma). This patient did not become aware of the tumor until her menstrual cycle stopped unexpectedly. She had had several successful pregnancies prior to this. In retrospect, it may be said that the abnormal nerve head was an early (i.e. congenital) warning which foreshadowed the development of the tumor.

The notion that the forces leading to development of a tumor in adult life are active during the earliest embryonic stages of development may seem surprising; however, this concept becomes more reasonable, as Taylor points out, when one considers that many of the tumors mentioned above are regarded as developmental in origin: they are due to the presence of pockets of embryonic tissue (embryonic rests) which undergo abnormal growth later in life.

Many people show situs inversus and other interesting nerve head anomalies, while only a few people develop tumors of the sort mentioned here. Nonetheless, this case should prompt at least a visual field assessment for patients with congenital nerve head anomalies.

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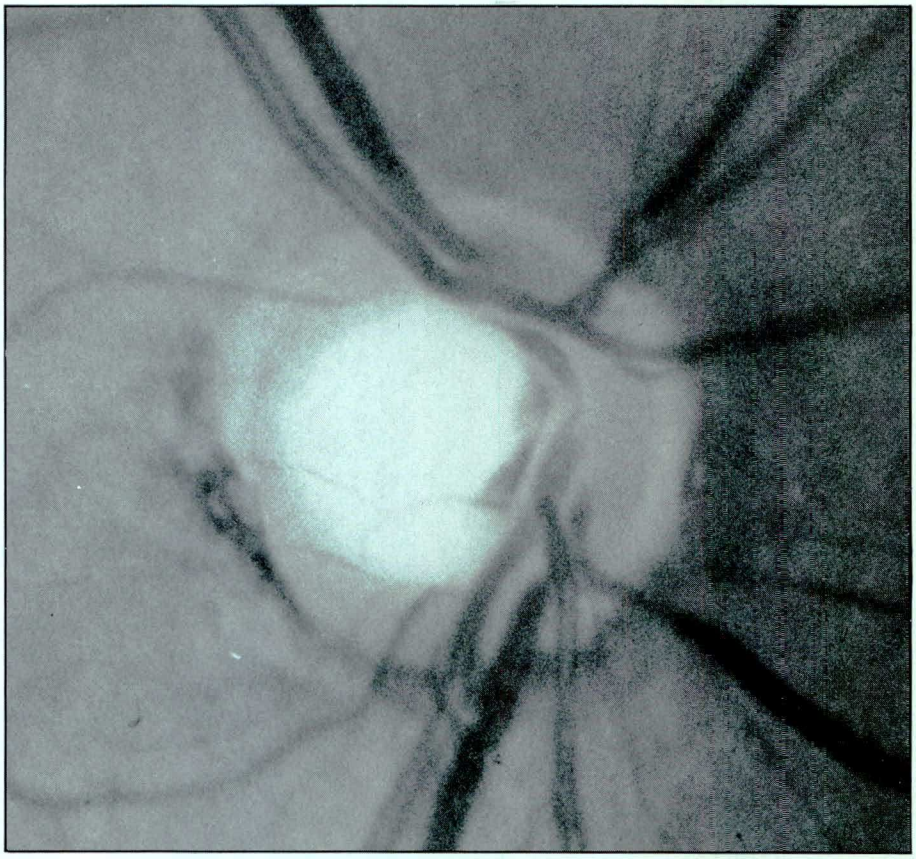


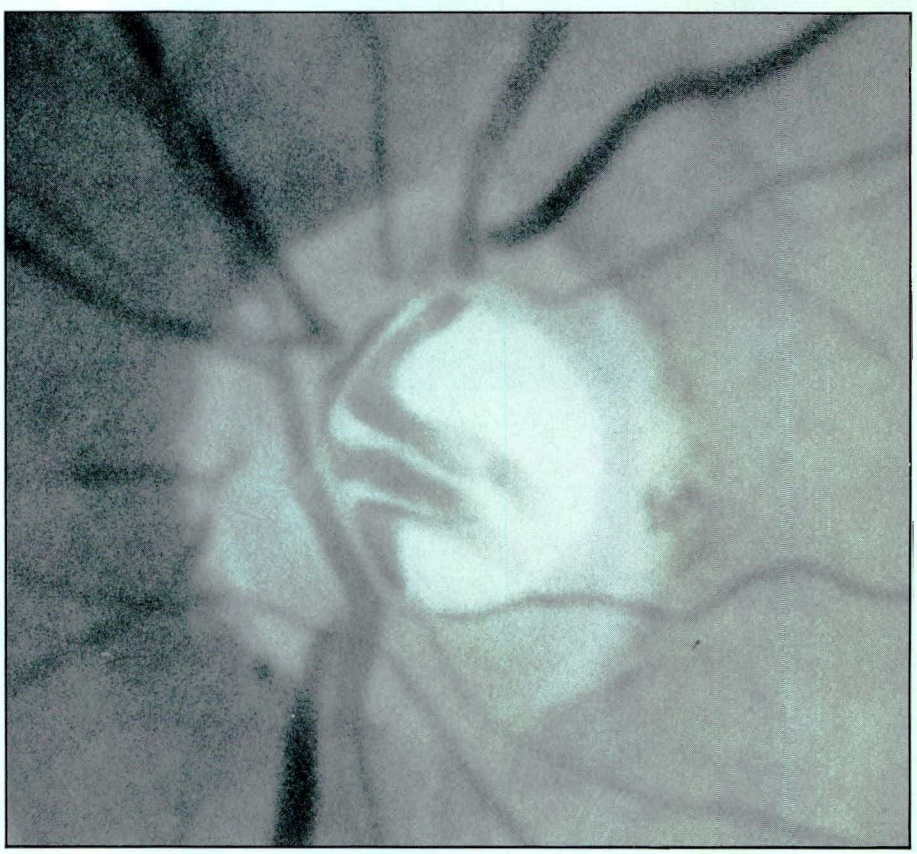
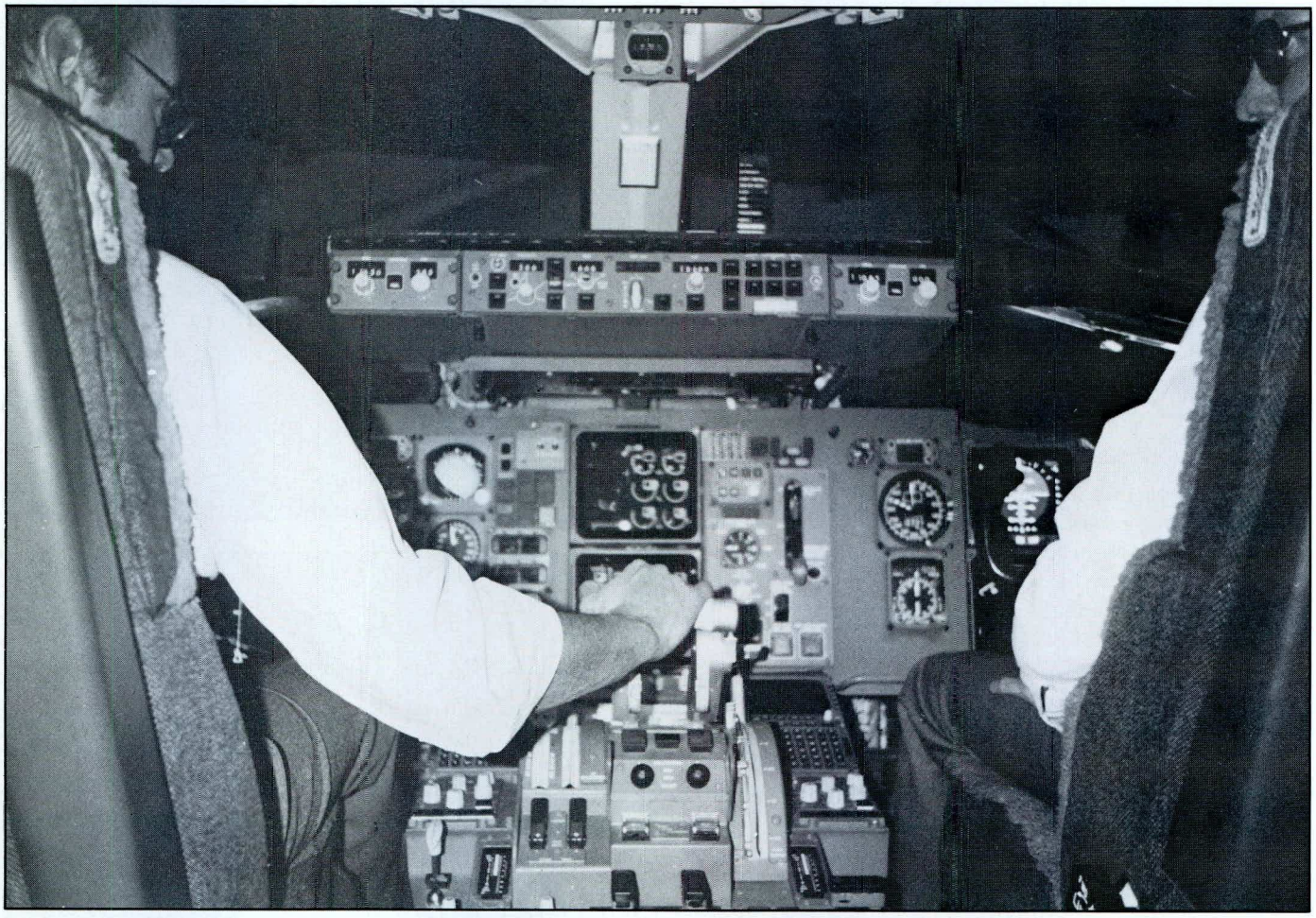


Fig. 3 (upper): right nerve head. Deep Elschnig II type with well-defined, unbroken neuroretinal rim. It was apparently this deep cupping which prompted referral by her GP for a glaucoma assessment.

Fig. 3 (lower): left nerve head. Central retinal vessels (as seen at bottom of cupping) appear to originate at the temporal side of the nerve head. This is typical of situs inversus. A deep Elschnig II type, also with well-defined (and unbroken) neuroretinal rim.

Aviation Vision and the Optometrist

L.G. Hart *



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Abstract

With new Federal legislation requiring the pilot to identify himself or herself as a pilot to the examining optometrist, it is important for the optometrist to have some knowledge of the flight environment.

Basic optometric techniques such as visual acuity measurements, depth perception, colour vision, etc. are discussed and related to the working world of the aviator.

Future cockpit designs and their affects relative to the visual systems are also discussed.

As has been reported in a previous issue of the CJO (Volume 47, No. 3, September, 1985), Bill C-36 requires flight personnel to identify themselves as such to the eyecare professional. It is also the responsibility of the examining doctor to notify the pilot of any visual disorder which might affect his flight performance and to request his grounding until the problem is cleared up.

It is with this in mind that this article will review basic optometric techniques related to the flight environment, will give an overview of the flight environment itself and what changes in the cockpit we should expect in the future. This paper will give the optometrist a greater understanding of the visual world of the pilot allowing the eyecare practitioner to better care for the needs of his/her pilot patients.

Basic optometric techniques remain the same in the refraction of the pilot or pilot candidate. It is the interpretation of results based on a knowledge of the flight environment which is the important variable.

The visual world of the pilot consists of an "office area" (inside the cockpit) and an external area, (that area outside the cockpit). The "reading" distances (inside the cockpit) range from 37.5 cm (15") to 90 cm (36") with various degrees of lighting and sources of lighting. The external environment ranges from 75 cm (30") to infinity, also with varying degrees of lighting.

Not only is clear, sharp, comfortable vision a requirement for each environment, but the pilot must be able to move easily between the environments. While this is

accomplished without problem by the youthful pilot, it increases in difficulty as the individual ages.

It is important for the practitioner to determine the type of aircraft flown as the cockpit environment can be drastically different from one aircraft to another. For example, the wide window area and dial complexity on the flight deck of an L-1011 contrasts sharply with the enclosed cockpit space of the Cessna 150. The pilot is able to adjust seat height etc. so that his working distance will remain approximately the same. Lighting can be adjusted by using thermostats; glare shields help in reducing outside glare.

The external environment presents a more complex picture. It can range all the way from the blackness of an overcast night with no visual reference points whatsoever to the brightness encountered when flying into a setting sun above the clouds. Lightning flashes will also add a deleterious variable to dark adaptation.

The effect of night myopia, plus the close supervision of lighted dials will serve to reduce distance acuity. Bright sunlight will produce visual fatigue and thus lower a pilot's visual efficiency. All of the above factors can be experienced by a flight crew when flying through many time zones, often during a single flight.

All of these factors must be kept in mind when the pilot patient comes for a visual consultation. It is not only important to examine the patient but the practitioner must be able to advise him or her about the problems which might be encountered in flight.

Visual Acuity

Snellen testing alone is a poor predictor of overall visual ability[8]. The standard acuity test typically uses high contrast optotypes at a high level of illumination to determine only the smallest detail that can be seen. However, a person's visual capabilities are not limited to high contrast conditions. The visual world of the pilot contains objects of varying sizes and often poor contrasts that must be viewed under both ideal and poor viewing conditions. Good vision requires sensitivity to contrast.

In one air force test, using a simulator[8], a group of pilots with the same visual acuity were asked to locate a plane parked in the middle of a runway. Some pilots actually saw the plane the equivalent of a full mile sooner than other pilots!

The eye must not only bring an object into focus, it must be able to separate that object from its background. The contrast sensitivity test determines an observer's ability to see a wide range of object sizes under different contrast levels[13].

Depth Perception

It has been argued that good depth perception is not necessarily an important factor for safely flying an airplane[4]. Therefore, more people with suppression and monocular amblyopia are being certified to fly. As we will see, however, these conditions will become grounds for failing the flight physical when new cockpit designs become operational.

The helicopter pilot must have perfect depth perception because most helicopter flight is low level and contour flying requires excellent depth perception. Under these conditions, excellent hand-eye coordination is also essential.

Phorias

International Civil Aviation Organization (ICAO) regulations stipulate 6 eso - 6 exo and 1D hyperphoria as the outer limits for flight acceptance[14]. Of prime concern to the practitioner when phoria testing is the possibility of diplopia's occurring when the pilot is confronted with stressful low level illumination. This must be kept in mind by the eyecare professional as he or she goes through the battery of muscle balance tests.

Peripheral Tests

Good peripheral vision is an essential ingredient for safe landings. Awareness of our peripheral world as we look ahead locates our position in space in flying just as it does in driving a car. The sudden appearance of an object in our periphery should trigger an immediate response in order either to avoid a collision or to set in motion emergency procedures.

(When testing flying personnel, the author routinely uses the 1 mm white target in his perimeter. Target movement is faster than that utilized for the ordinary patient as he also likes to evaluate the speed of response.)

Colour Vision

The proper discrimination of colours is also important for safe flight. Taxi runways, with all their exits and entrances, are lined with blue lights while the take-off and landing runways are lined with white lights. Most modern airports also line the center of the main runway with white lights set in concrete.

It has happened often that pilots have landed or tried to take off on taxi runways. Full colour discrimination, therefore, is a must.

Within the cockpit, coloured lights and, in particular, red ones are used to attract the pilot's attention as emergency indicators and as landing aids in the Vertical Approach Slope Indicator (VASI) system. For example, in the two-bar VASI, a high approach will be seen with both bars white, while a low approach presents both bars red, with red or white being the proper profile.

Cathode Ray Tube (CRT) sets in Boeing 767 generation aircraft have varying shadings of colour. As many as four sets can be in use at one time, all using different colour combinations (Fig. 1). This can contribute to visual fatigue and ways to counteract this must be discussed with the pilot.

Dark Adaptation

With the adaptometer, practitioners test the eyes' ability to recover from glare. Speed of reaction is also measured. But the adaptometer can be used in another way. One of the difficult decisions we have to make with a pilot is when to prescribe at distance. As stated previously, using the acuity chart is not enough. The pilot who measures 6/6 or 6/9 in the office and needs, for example, a +1.00 or a +1.25 for 6/6 acuity will not be able to wear the R_x in flight. They usually complain of a reduction in distance acuity. This is probably due to the effects of space and/or night myopia. The author, in practice, reduces the final prescription by 0.50 diopters. To determine the most comfortable final R_x he will test the pilot in the adaptometer with the full correction found and then with the 0.50 D. reduction and then prescribe the lenses which give the best results.

Refraction

Plus or minus three diopters of power has been designated by ICAO regulations as the outer limits of acceptable ametropia[14].

People interested in aviation as a career, whose refractive error might surpass these limits in the future, must be advised about this before they make aviation their life's work. Hopefully, in the future, this regulation will be modified because the uncorrected visual acuity which these range of powers represent

will vary with age, media and macular conditions.

Cockpit distances

The single most important factor the examining optometrist must remember when examining a pilot (especially the presbyopic one) is the varying visual distances used by the pilot. It is essential for the practitioner to know the type of aircraft flown and what level of command functions are carried out by the patient.

Even knowing the cockpit layouts of most commercial aircraft flown, the author usually asks the pilot to measure the distance from his or her eyes to the major instrument areas, as each pilot naturally likes to set the seat in the most comfortable position. Most aircraft have positioning mechanisms which allow them to set the seat precisely at the position most comfortable for the individual pilot.

The distances from the pilot's eye to the instruments in the overhead panel in the Boeing 767 ranges from 37 cm (15'') to 65 cm (26''). Most pilots will sit at least 75 cm (30'') from the front panel which puts them at least 90 cm (36'') from the dials on the 1st officer's side. The 2nd officer has an engineering board to look at on the bulkhead directly in front which is probably 53 cm (21'') away and he or she will also look at the dials on the front panels which could be 100 cm (40'') +/− away.

Some aircraft have overhead dials which don't necessarily have to be read, eg. the Lockheed L-1011, while others like those in the 767 must be read. This will necessitate the placement of a bifocal in the upper portion of the lens.

In the author's opinion, the lineless bifocal will become more and more popular with the flying fraternity. Tests[2] are currently underway to determine how well they work in the cockpit. In his personal experience, the author has had varying results equal to those found in the normal population. Some claim to love them, others cannot adapt. Again it takes that special knowledge of one's patient which will determine the type of bifocal used. The switching of focal distances must be performed quickly, efficiently and comfortably. This makes the fitting of bifocals of extreme importance. In an emergency situation, a pilot cannot be hampered by a poorly fitted or designed optical correction.

Sunglasses

The tinted lens of choice is the gray #4 lens[4]. In this author's experience, it provides the best glare protection without altering the colour of the outside world and will probably maintain its popularity for years to come.

The varigray lens generally doesn't react well in the cockpit as enough ultraviolet light to affect the change will not penetrate the windshield.

Polaroid lenses will pick up windscreen striations and produce visual fatigue and some visual distortion.

When bifocals are worn, the author generally will recommend a tint in the upper portion of the lens, gray #4, leaving the bifocal area clear. This enables the pilot to read dials without having to use dial illumination in daytime.

Visual Enhancement

For years, professional auto racing drivers have made use of various forms of visual training to enhance their reflexes and thus their driving skills. More recently, a whole new specialty, sports vision, has evolved because of the optometrists' ability to upgrade an athlete's visual skills.

These same sports vision enhancement techniques can be used to help the aging pilot. The ability to react to outside visual stimuli and cockpit emergencies with speed and accuracy is essential for safe flight.

Minimums have been developed which can be used to give an approximation of the speed with which an action can be performed under optimum conditions. As the pilot ages, it is reasonable to expect that under stress situations he or she will react below these limits. Reaction time, however, can be enhanced with training. This can be done in the simulator and is the subject of a future paper under development by the author.

Future Cockpit Designs and their Relationship to the Visual System

The appearance of electronic flight displays are already changing the design and appearance of aircraft cockpits. Traditionally, dials display such parameters as speed, direction, altitude, etc. The Electronic Flight Instrument System, however, displays all this information on only a few cathode-ray tubes. This system already exists in the Boeing 767 and will be even more sophisticated in the advanced A 320 passenger transport.

Already in use in military aircraft, commercially we will soon have sophisticated computer-graphics systems which will collate data from on-board sensors and digital terrain maps to give a realistic three-dimensional view of the environment around the airplane[11].

A Heads-Up Display system will allow the pilot to keep his or her eyes fixed ''up front'' while still allowing other instrument information to be read as well. The information is displayed at eye level on a transparent screen (called a combiner) mounted on top of the console[5].

This Heads-Up Display unit is already being miniaturized and moved onto the pilot's helmet for use in fighter aircraft, thus widening the pilot's field of view. The screens are now closer to the eyes. The use of a combiner for each eye will present a pictoral stereoscopic display and it is only a matter of time before this system will be used in commercial aircraft.

Also scheduled for tests shortly is a system[11] which will simulate the scene outside the aircraft as well as show images of communications and navigation-control panels. The pilot will be capable of interacting with the display by issuing voice commands or pointing. This system consists of a computer linked to two small CRT's mounted on the pilot's helmet. An optical system actually projects the CRT's screen images onto the pilot's eyes to create a stereoscopic image. Sensors mounted on the helmet will enable the computer to plot the position of the pilot's head and thereby update the simulated scene accordingly.

Conclusion

Almost daily, practitioners have patients who use VDT's on the job complain about the effects experienced during CRT use. Granted, many of the complaints may be exaggerated but there are legitimate symptoms. Dry, burning eyes, headaches and after-image complaints do occur after prolonged use of the CRT set. These symptoms can be alleviated by frequently looking away from the set, work breaks, and reducing glare and reflections from the screen.

These problems, however, are not so easily solved in flight situations. Constant monitoring of the CRT screen will

be essential for safe flight and if the screen or unit is placed very close to the eye, the pilot will be unable to find relief. The visual effects will have to be carefully monitored. In the meantime, dry eye symptoms which are common in the cockpit will have to be alleviated with artificial tears and/or cold compresses.

The use of contact lenses in the cockpit has been dealt with in other articles [1, 3, 6, 7, 9, 10, 12] and will not be discussed here. It is sufficient to note that, with the acceptance of the contact lens by the aviation community, optometrists will be sought out more often to deal with visual problems that arise in flight.

When aviation was in its infancy and "seat-of-the-pants" flying the "right stuff", good eyesight was prerequisite for safe flight. Technology, however, evolved the modern sophisticated flight deck with its load of instrumentation and multi-pilot systems. Monocular amblyopes and even one-eyed pilots are now being certified with no detrimental effects, up to the present time, being documented. With the advent of the futuristic cockpit with its multitude of CRT sets, Heads-up Display units and flight systems using stereoscopic optical devices, we will be forced again to choose aviators who have perfect vision.

As eye care professionals who will be dealing with more and more aviation personnel, optometrists must know the environment in which the pilot works so that we will be able to deal with the visual problems when he or she seeks consultation.

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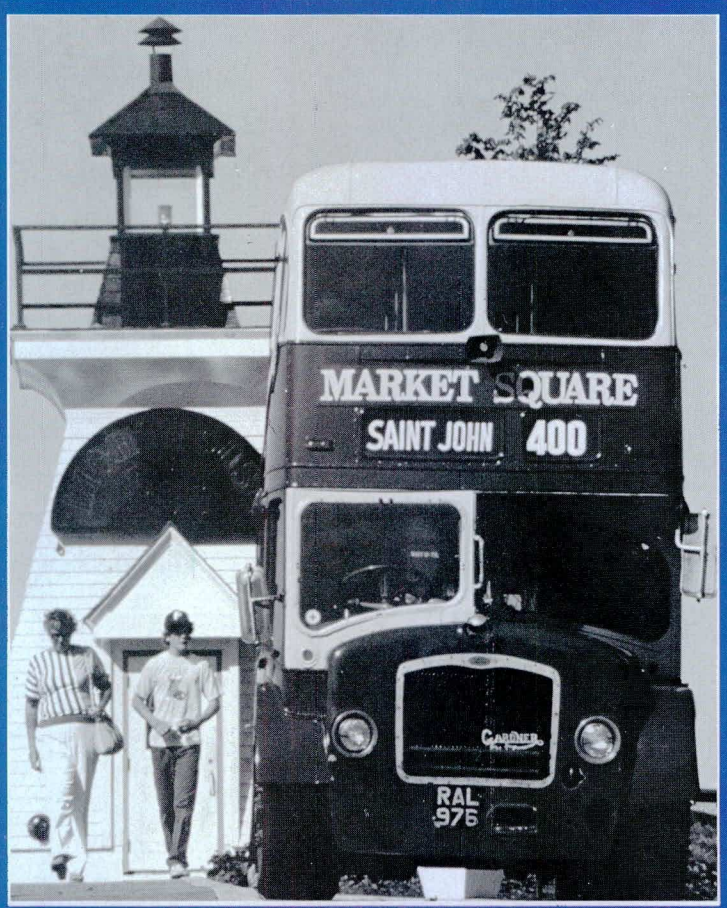

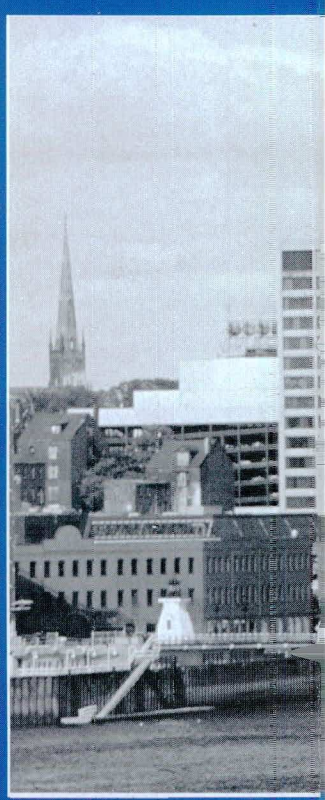

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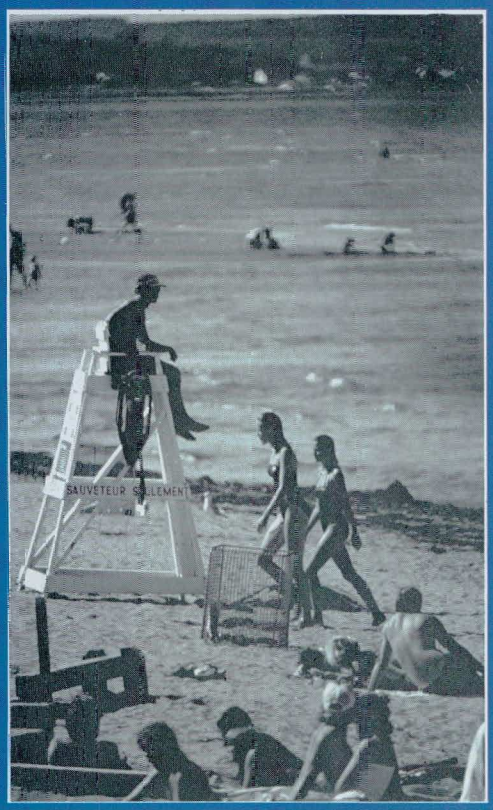

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À propos des formules d'inscription

Les formules d'inscription au congrès ont été envoyées par la poste à tous les membres de l'ACO. N'oubliez pas que le rabais accordé à ceux qui s'inscrivent tôt prend fin le 30 mai. Droits pour les inscriptions hâtives : Optométriste membre : 250 $; conjoint ou invité : 225 $; délégué stagiaire : 125 $; garderie : 45 $. Les fiches de réservation d'hôtel seront mises à la poste avec les reçus.

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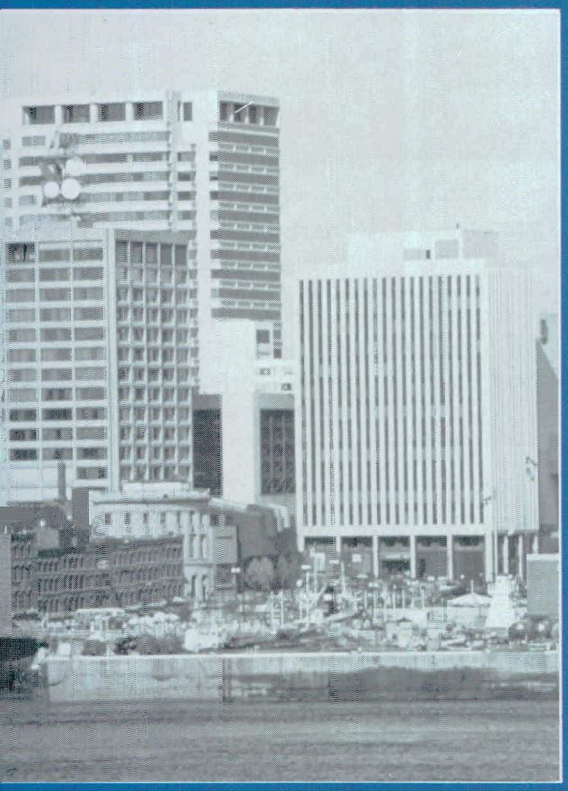

Evaluation of Soft Contact Lens Performance Using the Contrast Sensitivity Function

V. Timpano *

**O.D., Guelph, Ontario*

Introduction

The contrast sensitivity function (CSF) has been widely accepted as a measure of visual performance.[1] Its use as a diagnostic tool in contact lens work has been documented.[1,2,3,] Typically, the CSF has been used for the patient who presented with a complaint of blurred vision while wearing hydrogel contact lenses. Snellen visual acuity testing indicated little, or no decrease in vision. The contrast sensitivity test on the other hand, indicates a reduction in visual performance. Studies have shown that this reduction is usually due to a small residual refractive error, or to deposits on the lens.[1,4,5,] It sometimes happens that patients who have been wearing soft contact lenses successfully for some years require a new lens. Upon delivery of the new lens the patient does not achieve the expected performance.

Case Report

This is a report of a patient (D.B.) who had been wearing soft contact lenses successfully for five years. The most recent set of lenses had been fitted approximately one year ago. Her spectacle correction was −3.25 D.S. in each eye. The lenses were lost and new lenses of the same parameters were ordered. When the replacement lenses were delivered, Snellen visual acuity was the same in both eyes. The lenses were fitting properly, and the over-refraction was plano in each eye. The quality of the lenses appeared to be good. Within days D.B. informed us that her vision was blurred with her right eye. Since she was wearing identical lenses in each eye, she was instructed to wear the lenses on the opposite eyes. The difficulty was now transferred to D.E.'s left eye. It was suspected that the lens might be defective, but careful examination revealed no flaws. A replacement lens was ordered. The new lens gave similar results. At this point both patient and optometrist were frustrated. D.B. was refitted with a different type of hydrogel lens. With this set of lenses, equal vision was achieved both clinically, and from the patients[1] point of view.

Clinically, a problem still remained. How does one determine the effectiveness of a hydrogel lens when conventional testing is inconclusive? In this case contrast sensitivity testing proved to be helpful. CSF results, using the Vistech system, were compared with spectacles, with the set of lenses (A) with which she was experiencing monocular difficulty, and with the newer lenses (B). For the CSF testing D.B. wore the lens in question on her left eye. The results obtained are shown in Figures 1 and 2.

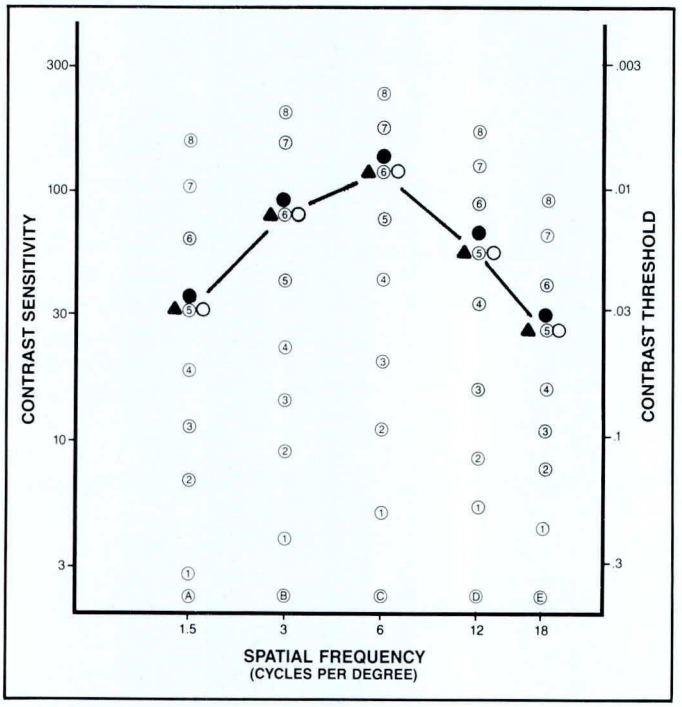

FIGURE I

— ● — SPECTACLES
— ▲ — LENS A
— o — LENS B

RIGHT EYE

Discussion

The results indicate a reduction in visual performance when wearing the lens in question. This reduction was only in the higher frequency end of the CSF. Reductions in the higher frequency portion of the CSF are indicative of a refractive, or optical problem of the eye, or of the lens being worn.[3,5,] Since measurement showed that the refractive power was correct, it was assumed, that the optical properties of the individual lens must not have been as good as the other lens.

Conclusion

Contrast sensitivity testing is being used to assist in the selection of the type of contact lens to be prescribed.[1] It is also used to determine if a patient is a good prospect for contact lens wear. In this particular case, it helped me to determine the optical performance of an individual hydrogel contact lens, where other test procedures provided inconclusive results.

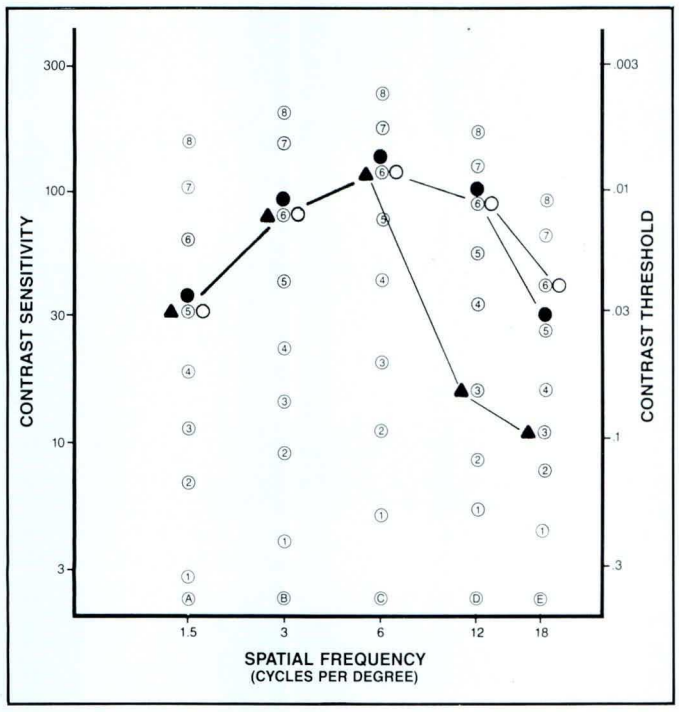

FIGURE 2

— ● — SPECTACLES
— ▲ — LENS A
— o — LENS B

LEFT EYE

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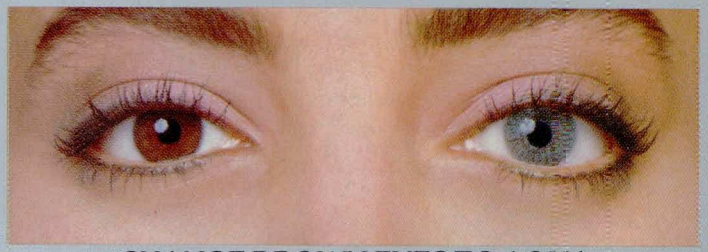

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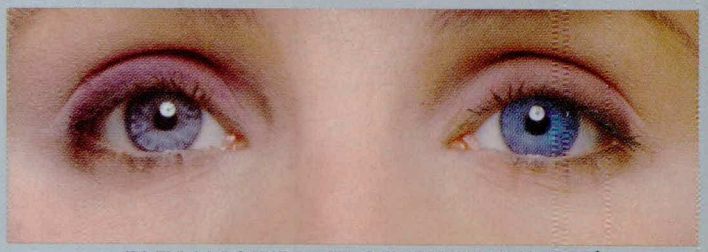

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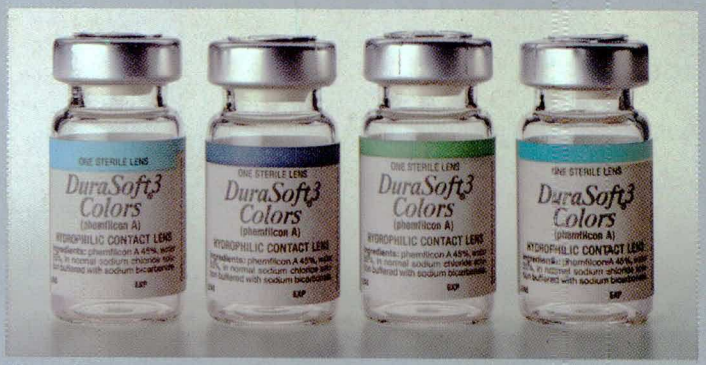

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RESEARCH/RECHERCHE

Headache

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Among patients consulting their optometrist headache is often the chief complaint.

Headaches are symptoms rather than disease entities. In most cases objective signs are few.

The differential diagnosis of headache is based to a large extent on an adequate health-illness history. The various features of the headache itself together with any associated signs and symptoms often provide a guide to the probable cause or indicate the need for additional tests.

Popular wisdom is not a reliable guide to probable cause, potential seriousness or suitable treatment.

It is hoped that the attached table will supplement the optometrist's clinical assessment and help to identify the type and probable cause of many headaches.

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Table 1

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Migraine, classical type, migraine simplex. (Classical migraine occurs in about 3% of the population and constitutes 10% to 20% of all migraines.) (About 80% have a positive family history. A hereditary factor is present in more than 50% of those with migraine; possibly autosomal dominant inheritance.) (See common migraine and other forms of migraine.)	Sick headache; pain is dull, boring, throbbing. The contracting and expanding images of the aura alternate at the same rate as the brain wave rhythm, about 10 per second. More than 20% of all migraine episodes are not preceded by an aura, but an aura is usual in classical migraine. (Scintillating scotomas have occurred in patients taking digitalis. They also notice changes in color vision, both effects suggest that the dose is excessive.)	Frontal, behind the eye; often on the same side in each episode. Usually begins unilaterally but in 33% spreads all over the head. Head pain begins on the side opposite from the scotoma in most cases. The headache develops gradually about 20 minutes after the aura begins. (Traction on the retina can produce a sensation of sparkling or flashing lights which might be confused with the scintillating scotoma of migraine.)	Commonly begins in the teen-age years. Headache-free interval between episodes is usually more than two weeks and attacks generally occur at regular intervals. Very rarely develop status migrainosus i.e. almost daily headache. If episodes occur very frequently suspect over-use of ergotamine or that the headache is not migraine or not migraine alone. Headache frequency or severity may increase in early pregnancy. Many women but not all become free of their migraine during the second and third trimester of pregnancy.	Twelve to twenty-four hours. Onset often in early morning and tends to ease off at the end of the day. Three stages: (a) prodromal (b) heaache, nausea (c) post headache. May end with sleep or after vomiting or after a sudden increase in mental or physical activity. Some discomfort lingers after the acute phase probably because neurokinin or other mediators are released in the area of tissue edema. Increased plasma levels of gamma aminobutyric acid and cyclic adenosine monophosphate. Headaches tend to become less severe after 50 years of age especially in women.	Transient depression of the electrical activity in the brain. Vaso-motor instability. Increased vascular permeability. Vasoconstriction of branches of the internal carotid causes cerebral ischemia and produces the aura. Followed by dilatation of branches of the external carotid which produces the pain. Serotonin released from platelets causes intracerebral vasoconstriction and extracerebral vasodilatation. Prostaglandins especially E_1 cause extracranial vasodilatation and thromboxane causes cerebral vasoconstriction. Stres or mild trauma can precipitate on attack.	Ergotamine tartrate if given in the aura stage i.e. within first half hour. Rebreathing from a paper bag so as to increase the carbon dioxide level in the blood may help in the aura stage. Some drugs are useful as prophylactics e.g. cyproheptadine, or methysergide, or beta blockers including propranolol. Stop birth control pills, stop smoking. For status migrainosus may need narcotics, ergotamine, steroids, sedatives, major tranquilizers. Calcium blockers help by stabilizing the tone of the blood vessels. Correct the refractive error and ocular muscle imbalance.	Prodromes consisting of depression, fatigue, hyperactivity or mood changes often precede the attack. Aura usually visual (teichopsia, fortification figures) precedes headache by 20 to 30 minutes. Closing the eyes does not cause the scintillating scotoma to disappear. Headache, nausea, vomiting, diarrhea, and polyuria follow. Unilateral paresthesias occur in about 30% and some have other neurological signs or symptoms, e.g. aphasia and mental clouding. Conjunctival hyperemia, photophobia, anisocoria, excess lacrimation, blur, transient scotomas (usually-homonymous) and an enlarged blind spot are often present. Micropsia and macropsia have been reported.

Table 2

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Migraine, common type. (Constitutes about 25% of all severe headaches and 70% to 85% of all migraines.) (Migraine is probably the second most common cause of severe headache.) (At least 10% of the population have some type of migraine.) (A family history of migraine is reported by 80% of those with migraine.) (Epilepsy occurs more frequently in families with migraine. Migraine occurs more frequently in families with epilepsy.) (A migraine-like headache occurs in 70% of patients undergoing renal dialysis but the mechanism for this headache is uncertain.) (There are many variants of migraine. See classical and other forms of migraine.)	Sick headache; pain is dull, boring, throbbing. The pounding headache often begins slowly during the REM period of sleep. Migraine sufferers have been described as tense, obsessional, meticulous, perfectinioistic, intelligent, neat, sensitive, ambitious, emotionally restrained etc. but there are many exceptions to this type of categorization. Migraineurs are also said to have a low basal metabolic rate and to be relatively infertile. Cerebral edema leads to the gastrointestinal disturbance.	Unilateral or bilateral, anywhere in head often in the temple or frontal area but can alternate beginning on one side one time and on the other side in the next episode. Often seems to be all over the head not just unilateral.	Usually begins in the teen-age years. Occurs once every two weeks or less often. Headache episodes occur at less regular intervals than in classical migraine. Migraine is slightly more common in women than in men. In women with migraine 60% of the attacks occur in association with menstruation. Headache attacks tend to decrease in frequency after 50 years of age especially in women. Migraine usually diminishes after the menopause but rarely it worsens. Migraine almost never begins after 40 years of age.	Twelve to twenty-four or more hours. May awaken the patient in the morning. Many patients feel particularly well or energetic just before an attack. Pain-free interval between episodes. Locally released chemical substances lower the pain threshold near the blood vessels so some discomfort lingers after the acute phase. The headache usually ends with sleep.	Altered neurogenic activity or vasomotor instability. Increased amplitude of pulsation of cranial vessels. Studies show no changes in cerebral blood flow before, during or after an attack. Typically migraine requires a genetic predisposition, a specific personality pattern and a triggering event. Precipitating factors include: fatigue, trauma, stress, estrogens (the pill), "eyestrain", low-tension glaucoma, hunger, flashing lights, noise, and certain odors. During an attack the blood has less endorphins and 50% less serotonin but more platelet abnormalities.	Ergotamine tartrate if given early. Some drugs are useful as prophylactics e.g. cyproheptadine, methysergide, or propranolol and other beta blockers. Pressure on the common carotid eases the pain transiently. Stop birth control pills. The migraine sufferer seeks a quiet dark room and lies down with a cold cloth over the eyes. Migraine headaches often disappear during the middle trimester of pregnancy. Migraine headaches usually diminish with major illnesses or surgery. Correct the refractive error and ocular muscle imbalance.	Drowsiness or mood changes often precede the attack. Generally no visual aura but may have vague, constitutional disturbances hours or days before which the patient learns to interpret as a warning. Nausea, vomiting, light headedness, and polyuria accompany the headache. A few suffer amnesia, confusion, or even psychoses. Most notice fluid retention before the attack and 20% have diarrhea during the episode. Conjunctival hyperemia, photophobia, scotomas, and rarely vasospasm of retinal arterioles and blurred vision. A transient Horner syndrome sometimes appears. Migraine patients are sensitive to lights, sounds and smells and more apt to notice spatial disorientation when adapting to new glasses.
Ophthalmic migraine, retinal migraine, sometimes called ocular migraine, occipital migraine, or even senile migraine equivalent. (Do not confuse with ophthalmoplegic migraine or with Roy's ocular migraine.) (In some abortive forms the visual aura is not followed by a headache, this may be called acephalic migraine).	Unilateral visual impairment precedes the headache. (Jagged figures, Moore's lightning streaks, are seen by some elderly people with degenerative changes in their vitreous. These vertical streaks can be confused with the fortification figures of migraine.)	Periorbital, frontal, behind the eyes. Unilateral or bilateral.	Rare but some have repeated episodes. May develop in older patients who have had migraine for years or appear in younger patients with no family history of migraine.	About 30 minutes. (Do not confuse with transient ischemic attacks which are mostly due to emboli. TIAs are monocular events which last for only a few minutes.)	Vascular causes probably account for 80% of all chronic head-pain complaints. If the disturbance affects vessels supplying the anterior portion of the visual pathway the visual phenomena are monocular. Vasoconstriction involving the vessels supplying the calcarine cortex causes binocular visual effects which do not disappear when the eyes are closed.	Propranolol is effective. Be sure no other cause exists then correct the refractive error and ocular muscle imbalance. (Retinal detachment can cause the eye to see sparks, shooting stars, a fountain of spots, lights or colors which might be confused with the aura of migraine.)	Young adult females. Unilateral retinal dysfunction occurs especially during the aura phase. Scintillating scotomas but no fortification figure. Sometimes have hemorrhages in their conjunctiva, retina or rarely in the vitreous; ischemic papillitis. Retinal hypoxia causes transient loss of vision lasting up to 10 minutes. Eventually pigmentary changes appear in the retina.

Table 3

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Roy's ocular migraine, latent vertical phoria syndrome. (This less well known type was described by Raymond Roy.) (Do not confuse with ophthalmic migraine or ophthalmoplegic migraine.) (See muscle contraction headache.)	Headache may be mild, or severe. A pulling feeling in the eyes may precede the headache.	Unilateral or bilateral, anywhere in the head and almost always extends into the neck muscles.	Daily in afternoon or evening but in a few cases wakens the patient in the early morning.	Chronic, usually manifests before patient is 20 years old. Often these patients have sought help from many different practitioners. Migraine of some type accounts for nearly 33% of all lost work days due to sickness.	Latent hyperphoria, May have hypertophthalmia i.e. one eye is anatomically higher in the head than the other eye. Develop postcervical tension from head tilt. Stress results from the induced hyperphoria. May be inherited as a dominant characteristic.	Be sure no organic cause exists. Uncovering and correcting the hyperphoria. The hyperphoria can be uncovered by the monocular occlusion technique of Marlow, Jacques, and Charnwood. Prescribe absorptive lenses and base-in prism if necessary.	Chronic fatigue, nausea, motion sickness, torticollis, stiff neck, nervousness, depression, insomnia. Severe photophobia; tend to suppress or close one eye, blink frequently. Poor stereopsis, poor readers, subnormal amplitude of accommodation, transient scotomas.
Abdominal migraine, juvenile migraine, cyclical vomiting, pediatric migraine, childhood migraine, cyclic infantile colic. (Compare with other types of migraine.)	Abdominal discomfort. Abdominal symptoms are more prominent than headache until about ten years of age then the throbbing paroxysmal headache develops in the second decade. Sixty percent of adults with migraine report that they had motion sickness during childhood.	Pain in abdomen in childhood but later in life manifests as headache. However, some adults have episodes of abdominal symptoms even into middle age.	Abdominal symptoms begin on the average about the age of six years. Headache begins after ten years of age.	Each episode lasts 6 to 12 hours. Cyclical vomiting usually disappears by 8 to 10 years of age but is followed by development of headache episodes in the teen age years.	Familial tendency. Childhood migraine may be secondary to or confused with systemic conditions including hypoglycemia, anemia, upper respiratory tract infections, fever, meningitis.	ASA, rest and sleep. Rarely ergotamine at half the adult dose for a child over the age of 6 years, or methysergide 2 to 4 mg a day. Cyproheptadine 4 to 8 mg at bedtime. Some respond to anticonvulsant drugs e.g. phenobarbital 30 mg t.i.d. but not to ergotamine tartrate. Correct the refractive error and the ocular muscle imbalance.	Affects more boys than girls. Affected children often have motion sickness commonly called car sickness. Some have a visual aura, or dizziness, most have nausea and cyclical vomiting. In a few cases the child becomes confused during the episode.

Table 4

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Dietary migraine and allergic migraine. These are really two separate types of migraine. (See toxic and infectious headaches.)	Headache usually with nausea and visual problems. Specific foods precipitate an attack in 30% of those with migraine. In the Chinese restaurant syndrome due to monosodium glutamate (MSG) those affected have a transient episode of throbbing headache with light headedness and increased muscle tension in jaw, neck and shoulders.	Bilateral or rarely unilateral. Mostly frontal but can extend to the vertex or occiput. Flushing of the skin of the face and chest occurs.	If a specific food is responsible the problem appears each time that food is ingested. Seasonal in those cases where some forms of allergy are associated. Up to 25% of the population may be sensitive to MSG.	Allergic type may be worst when patient first awakens.	Exposure to specific foods but much disagreement exists as to cause. Commonest causes are wheat, citrus fruits (octopamine), eggs, nuts, meats, yeast, nitrites, nitrates, alcohol, red wine, aged cheese, milk, seafoods, pickled herring, or ingestion of other tyramine-containing foods or the beta phenylethylamine in chocolate. Monosodium glutamate and nitrates act as vasodilators. Fasting or hypoglycemia may be factors in some patients.	Appropriate diet will prevent the headache in 30 to 80% of these patients. Be wary of food faddists or self-appointed nutritionists. Clonidine is often helpful if a rise in blood pressure is involved. Correct the refractive error and ocular muscle imbalance.	Be sure there is no organic cause. Be wary of placebo effects. Migraine attacks may alternate with episodes of urticaria, asthma or angioneurotic edema. Susceptible patients often have have nasal congestion, rhinorrhea, sneezing, visual problems, photophobia and conjunctival hyperemia. Recent studies indicate that MSG may not be solely responsible for the skin flush.
Complicated migraine, hemiplegic migraine, hemisensory migraine, aphasic migraine, ophthalmoplegic migraine, periodic oculomotor paralysis, Mobius's disease. (Probably accounts for 1% of migraines.) (Shows an inherited tendency, autosomal dominant.) (See basilar artery migraine, ischemic cerebrovascular disease, retinal migraine, subarachnoid hemorrhage and the Tolosa-Hunt syndrome.)	The abrupt onset, dull, migraine-type headache usually develops before the ophthalmoplegia or other forms of motor or sensory dysfunction. The headache in most other types of migraine does not begin abruptly. Ophthalmoplegic migraine is rare but is considered to be a more severe or ominous variant of classical migraine.	Unilateral ache above or behind one eye. Headache plus numbness in limbs or face or paralysis of certain muscles. Symptoms of motor or sensory dysfunction. The weakness and sensory disturbances can be unilateral or bilateral, and are mostly ipsilateral to the headache. Unilateral paresthesias occur in 30% of migraineurs.	This rare form of periodic migraine tends to be familial and probably begins in childhood, but is most likely to be seen in the young adult. Usually have irregularly recurring episodes over many years. The interval between attacks can be as long as five years.	Each episode lasts for only a few hours. All neurological signs usually clear within an hour if no organic lesion is present. Third nerve palsy of this type in a child usually clears in six weeks. Incomplete recovery is possible in hemiplegic migraine and the condition is then called complicated migraine and provides more cause for concern.	Edematous arterial walls impinge on nearby nerves. A predisposing cause seems to be cerebral ischemia due to focal arterial spasm. Suspect an aneurysm until this is ruled out. Ipsilateral herniation of the hippocampal gyrus of the temporal lobe through the incisura tentorii is one cause. There is an elevated level of gamma-aminobutyric acid in the cerebrospinal fluid and the platelets are hyperaggregable.	Be sure there is no organic cause. A CT scan often provides useful diagnostic information but cannot reveal very small aneurysms. May need an angiogram especially if epilepsy occurs too but the risks associated with this procedure are even greater in these patients. Prednisone helps if administered promptly. Correct the refractive error and ocular muscle imbalance.	Mostly young adults. No nausea. Ocular palsy occurs in 17% of all migraine sufferers and of these 80% involve the third nerve which is close to the internal carotid and middle cerebral arteries, or is compressed between the posterior cerebral and superior cerebellar arteries. Can affect cranial nerves III, IV, VI or any part of V. Transient speech defects or impairment of the sense of smell occur only rarely. Some have field losses, ptosis, diplopia, muscle weakness or paralysis, pupillary dysfunction. More than 33% have a fixed dilated pupil.

Table 5

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Cluster headache, Harris' periodic neuralgia, histaminic cephalalgia, migrainous neuralgia, Horton's syndrome, Bing's syndrome, Kunkle's cluster headache. (Cluster headaches affect 0.5% of the population and constitute up to 4% of the severe headaches. About 33% have a family history of similar headaches.) (See Charlin's syndrome, both may be called ciliary neuralgia.) (See Sluder's syndrome, both may be called sphenopalatine ganglion neuralgia.) (See carotidynia, Raeder's syndrome and chronic paroxysmal hemicrania.)	The abrupt onset headache is intense, boring, burning. Victim may cry, moan, yell, pace the floor, bang the head, even roll on the floor. More severe pain than in most migraine headaches. Occipital pain may be carried by CNIX and X and the upper cervical nerves. (An atypical cluster headache may be mild or severe, and last hours, days, or even persist for weeks. Some of these patients have hypertension, arteriosclerosis, or infections. Trauma is a cause of some headaches of this type.)	Unilateral near one temple. Pain seems to follow the distribution of the external carotid artery. Nearly always pain is on the same side of the head in each attack. When the pain is supraorbital the condition is referred to as the upper syndrome. In the lower syndrome the pain is infraorbital. The nervus intermedius (Wrisberg) a part of CNVII is the main conduit for cluster headaches, it carries parasympathetic fibers. (In the atypical type the pain follows the distribution of the trigeminal nerve, CNV.)	Attacks tend to develop late in the second decade of life. They have two to five episodes a day for several consecutive weeks, then a clear interval of months or years followed by another cluster of attacks. The mean remission period between clusters is one year. A few people have little or no pain-free interval between headache episodes so have atypical or chronic cluster headaches.	Most of these headache episodes last less than one hour. Often they awaken the patient from sleep between midnight and two a.m. They are associated with the REM period of sleep.	The upper syndrome is due to dilatation of the external carotid and its branches. The lower syndrome is due to dilatation of other large arteries e.g. internal carotid, vertebral, basilar. An "autonomic storm" involving visceromotor centers in the brain has been blamed. Most are smokers, and use alcohol. Worsened by alcohol ingestion or by lying down. Seasonal and allergic factors but few emotional ones have been reported. Is growth hormone related? Cluster headaches are more common in those with hazel irides.	Methysergide is a useful prophylactic. Ergotamine tartrate in low doses can also be useful prophylactically but unless given IV or as an aerosol it is too slow acting to treat an acute episode. Some use prednisone, oxygen, vasoconstrictors, indomethacin, or lithium carbonate. The pain is transiently eased by pressure on the ipsilateral temporal artery. Stop alcohol intake, stop smoking. Be sure it is not an episode of angleclosure glaucoma. Correct the refractive error and ocular muscle imbalance.	Mostly affects men 25 to 45 years of age. No prodromes, no trigger areas, no scotomata, no nausea. May have hyperalgesia of the face and scalp and tenderness over the dilated temporal artery. More risk of peptic ulcers. Said to be squarejawed, have coarse, rough, thick, facial skin and furrowed forehead. Have ipsilateral rhinorrhea and skin hyperemia, ipsilateral conjunctival hyperemia and chemosis. Edema around the eyes, increased lacrimation, sometimes miosis and ptosis. Often an ipsilateral, postganglionic Horner syndrome with the sweating mechanism intact. The small pupil is supersensitive to phenylephrine.

Chronic paroxysmal hemicrania is a variant of cluster headache.	Milder than cluster headache.	Unilateral.	More frequent episodes than are usual in cluster headache.	Episodic.	See cluster headache.	Indomethacin.	Rare, mostly affects young to middle-aged women.
Basilar artery migraine, vertebro-basilar migraine, Bickerstaff's migraine, basilar artery insufficiency syndrome, cramiocervical syndrome. (Over 40% of those with vertebro-basilar artery disease have headaches.) (See Ménière's disease, complicated migraine, post-traumatic headache, and ischemic cerebro-vascular disease.)	With occlusive disease of the vertebral-basilar system 50% develop a severe, throbbing headache. The visual disturbance is often followed by loss of consciousness.	Occipital and parietal; may have tender areas, stiff neck, back pain. May have facial numbness on one side and a motor loss on the other side of the body. An aneurysm of the basilar-vertebral vessels does not produce pain in the ocular region. The vertebro-basilar system supplies blood to the occipital cortex.	Episodic. Usual age of onset is the second or third decade. Often associated with menstruation but can occur in children. Transient ischemic attacks associated with vetebro-basilar insufficiency cause loss of muscle coordination, and homonymous hemianopia.	Brief paroxysms lasting 10 to 30 minutes. Persisting manifestations of these or similar symptoms suggest chronic vertebro-basilar insufficiency. Posterior cerebral insufficiency causes homonymous hemianopia with sparing of fixation. Basilar artery insufficiency causes dizziness, vertigo, drop attacks, diplopia and nystagmus but seldom causes death.	Ischemia of the reticular formation produced by spasm or occlusive disease of the basilar-vertebral arteries. Involvement of brainstem, pontine tegmentum, hypothalamus, thalamus, and occipital lobe. Can follow a whiplash injury or emotional stress. Can be worsened by turning head to one side. Complete thrombosis of the basilar artery can cause death.	Difficult to treat. Some patients are helped by anticonvulsants. Inhalation of 5% carbon dioxide has been recommended. Occlusion of the branches of the basilar artery which supply the pontine region produces the Raymond Céstan syndrome with quadriplegia, anesthesia and nystagmus.	Vasospasm is more apt to occur in young women. Some develop ataxia, tinnitus, vertigo, dysarthria, paresthesias, drop attacks, dysphagia, confusion as well as sensory and visual disturbances. Nausea and vomiting often occur and so can loss of consciousness or hemiplegia. Bilateral graying or blurring of vision, scotomas, bilateral homonymous hemianopia, usually lasting under 5 minutes. Vertical nystagmus, ptosis, transient diplopia, reduced accommodation, cortical blindness.

Table 6

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Quincke's angioneurotic edema. (Sometimes inherited as an autosomal dominant.) (See the Melkersson-Rosenthal syndrome.)	Headache.	Cerebral edema is responsible for the headache. Lid edema can cause ptosis.	Sudden onset in young patients.	Brief recurring episodes at irregular intervals but condition may reappear for years.	Cause obscure. Patients fail to synthesize an inhibitor of complement C_1 or produce an abnormal protein.		Edema of skin and mucous membranes. Can involve the respiratory and gastrointestinal tracts. Bradycardia, facial paralysis, deafness, mental confusion, loss of consciousness, paralysis of CNIII. Pupillary anomalies, uveal involvement and rarely papilledema.
Headache with hypertension. (High blood pressure causes less than 5% of all headaches.) (Only 50% of those with hypertension have headaches.) (Hypertension often accompanies arteriolar-sclerosis, diabetes mellitus, kidney disorders and toxemia of pregnancy.)	Headaches are dull, diffuse, throbbing and only rarely paroxysmal. Become severe when cerebral edema develops. A few report a sensation of a tight band. Headaches of this type are most likely to be reported with a pheochromocytoma or with eclampsia.	Generalized, unilateral, frontal, occipital or at vertex; often at base of skull. Poorly localized.	Headache is worst in early morning and peaks just before the patient gets up. Often awakens the patient. Some prefer to sleep in a sitting position or to elevate the head of their bed.	Tends to wear off during the morning after patient gets up thereby helping blood to drain from the large intracranial venous sinuses. Some patients have this type of headache for years. With a pheochromocytoma 80% have a paroxysmal headache in the early morning which persists for about 30 minutes.	Elevated blood pressure. Only when diastolic pressure reaches about 120 mmHg is headache almost sure to occur. Often have cardiac or renal disease. Worsened by straining, coughing or stooping. Some develop sustained muscle contraction too.	Diuretics, clonidine, and beta blockers to lower blood pressure; more exercise, less salt, stopping smoking, losing weight; also eased by rest and relaxation. Transiently eased by pressure on the common carotid.	Many people with elevated blood pressure have no symptoms. A few report dizziness, light headedness or tinnitus. Hypertension is the cause of 10% of the muscle palsies which occur in older patients. Often retinal signs, especially arteriolar constriction or retinopathy or in severe cases papilledema. A venous pulse is not visible on the disc if the intracranial pressure is elevated.
Headache with temporal arteritis, cranial arteritis, giant cell arteritis, also called Horton's syndrome, the Hutchinson-Horton-Magath-Brown syndrome or Rumbold's disease. (See cluster headache.) (Polymyalgia rheumatica is a related condition which has only rarely been responsible for loss of vision.)	Severe, burning, steady or throbbing headache occurs in some but not all patients with this condition. Burning pain is an unusual feature for a vascular type headache. Headache of recent onset in an older patient should suggest temporal arteritis as one possibility.	Temple area, unilateral or bilateral, sometimes occipital. Scalp tenderness. Temporal artery is tender, enlarged, and sensitive. Pain when chewing due to jaw claudication. Jaw pain, trouble swallowing.	Temporal arteritis or giant cell arteritis is a rare but serious condition. Pain is usually worse at night.	Persistent headache is usual but the condition tends to clear in less than two years. Evidence of temporal arteritis is found in 1/1000 patients at autopsy but many had no related symptoms.	An immunologic vasculitis. The temporal artery is usually but not always involved. An involved temporal artery has a pulse which is less evident even though the artery appears to be prominent. Headache made worse by lying down, eased by upright position. The ischemic optic neuropathy is the result of occlusion of the posterior ciliary arteries not occlusion of the central retinal artery.	A true ocular emergency requiring immediate treatment. Steroids are required promptly. Prednisone 40 to 60 mg a day then taper off after about six weeks. Headache worsened by exposure to cold. A biopsy may be needed to confirm the diagnosis and strangely has been said to give some relief itself. The arterial involvement is patchy so a biopsy can be misleading. Temporal arteritis can kill.	Mostly females over 60 years of age. Ipsilateral temporal artery red, swollen and tender in 50% of cases. Anorexia, weight loss, arthralgia, elevated sedimentation rate, fever, weakness, malaise, fatigue, photophobia. Diplopia occurs in 10 to 15% and can be the result of damage to CNIII or VI. Temporal arteritis patients can lose vision in 1 to 4 weeks after onset of the disease. About 50% have severe visual loss which can become bilateral and permanent.

Table 7

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with ischemic cerebrovascular disease. (About 30% have headaches.) (See basilar artery migraine, complicated migraine, carotid artery syndrome, stroke headache and carotidynia).	Some are throbbing, some are steady. Intensity varies but is often mild. If a visual hallucination occurs it will disappear when the eyes are closed if the circulation to the eye is impaired but not if the basilar-vertebral arteries are affected.	Frontal and ipsilateral to the affected vessel if the carotid is involved. Fleeting weakness of an arm or leg or transient sensory disturbances can occur with or without a vision disturbance.	Headache may precede or accompany the ischemic condition. Transient ischemic attacks usually indicate cerebrovascular disease, emboli, or impaired circulation but can be due to hematological disorders or elevated intraocular pressure.	Minutes, hours or days. Transient dimming or graying of vision, if it occurs, lasts for not over 10 minutes and can occur with no pain and no other motor or sensory disturbance.	Atherothrombiotic disease affecting the internal carotids or the vertebral arteries or their branches causes headache in 30% of those affected. If the carotid is affected look for a prominent pulse in the temporal artery.	Ophthalmodynamometry is used to aid in the diagnosis. Some can be helped by surgery, endarterectomy. Risk of stroke is high. Listen for a carotid bruit. Idiopathic orthostatic hypotension does not usually cause a headache.	Do not confuse these unilateral transient ischemic attacks (TIAs) with the aura of migraine. With a TIA some have slowly pulsating photopsias and develop confusion without loss of consciousness. The eye on the side of the affected carotid may show cholesterol crystals in retinal vessels or develop occlusion of arteries or veins.
Headache with a "stroke". Strokes are often called cerebrovascular accidents. (See carotid artery syndrome and ischemic cerebrovascular disease.)	Severe, sudden onset headache occurs in 25% of strokes. The lenticular striate artery, a branch of the middle cerebral artery has been called the artery of cerebral apoplexy.	Thrombosis of the internal carotid artery produces a sudden, unilateral headache. A thrombotic stroke affecting the internal capsule causes extensive paralysis but little intellectual impairment. A thrombotic lesion near the cerebral cortex causes less paralysis but always causes more intellectual impairment.	Occlusion of the anterior cerebral artery causes hemiplegia and hemiparesthesia of the contralateral leg. Occlusion of the middle cerebral artery can cause contralateral hemiplegia, hemiparesthesia of the contralateral face, head or arm, and homonymous hemianopia with splitting of fixation and usually anisocoria. The lower quadrants of the visual field are most likely to be affected.	Occlusion of the posterior cerebral artery causes a transient ischemic attack and homonymous hemianopia with sparing of fixation and sometimes causes an aura but seldom death.	About 50% are due to thrombotic occlusion of vessels, about 33% are due to hemorrhage and 10% are due to embolism.	Treatment is controversial. Permanent neurological damage usually follows and one third die soon after the episode.	Stiff neck, continuous vomiting, contralateral paralysis of one side of the body, speech disorders, blood in the spinal fluid, hemiparesthesia of the contralateral face, hand and arm.

Table 8

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with the carotid artery syndrome, vascular insufficiency syndrome. (See stroke, carotidynia, ischemic cerebrovascular disease, ophthalmoplegic migraine and the Tolosa-Hunt syndrome.)	Severe unilateral headache with transient weakness or loss of sensation. Sensory or motor defects develop on the contralateral side. If the dominant hemisphere is involved speech will be impaired. If a visual hallucination occurs it will not be seen when the eyes are closed. Ophthalmic manifestations occur in 40% of cases.	Pain is facial and ocular. Aneurysms of the internal carotid cause unilateral, frontal or orbital headache. Such an aneurysm occurs more commonly in females and generally causes paralysis of CNIII.	Carotid artery stenosis causes transient ischemic attacks, unilateral hemiplegia, pain due to ischemia. Impaired circulation in the ophthalmic artery causes amaurosis fugax, ischemic optic neuropathy, and sometimes serious interruption of blood flow in the short posterior ciliary arteries or in the central retinal artery as well as rubeosis iridis and unilateral cataract.	Cavernous sinus thrombosis produces orbital effects i.e. exophthalmos, edema of lids and as well as papilledema, reduced acuity and impaired pupillary reflexes.	Ischemia. Frequently due to atherosclerosis.	A carotid bruit suggests carotid stenosis. An ocular bruit suggests a fistula. Surgery helps some of those affected, usually fistulas can be occluded. Ophthalmodynamometry can help to indicate the responsible artery.	Mostly affects males over 50 years of age. Dizzy, confused, may have a seizure. The amaurosis fugax attacks last for 5 to 15 minutes and are unilateral. Some have transient, monocular, contralateral loss of vision, hypoxic retinopathy, anterior uveitis, chemosis. Look for cholesterol crystals in retinal vessels (Hollenhorst plaques), raised IOP, pulsating exophthalmos, diplopia, paresis of lateral rectus. Pupil may become non reactive or be a Horner pupil.
Head pain with carotidynia. (See cluster headache, Charlin's syndrome, Sluder's syndrome, carotid artery syndrome and ischemic cerebrovascular disease.)	Usually a dull, throbbing headache but not always present. Not as severe as migraine. A similar ache develops within 48 hours after carotid endarterectomy.	Unilateral, neck pain in region of the common carotid radiating to face and ear. Cranial nerve V. Trigger area in floor of mouth.	When present varies in intensity.	Chronic for many years but pain appears in episodes and persists for 24 hours.	Caused by painful dilatation of the common carotid artery. Supranormal pulse in temporal artery. Worsened by pressing on the artery or by yawning, coughing or sneezing.	Steroids usually help. Ergotamine and propranolol have been used prophylactically. Listen for a carotid bruit.	Tends to affect women aged 40 to 50 years. Patients with atherosclerosis of their internal carotid artery are apt to suffer transient ischemic attacks or a stroke.

Cavernous sinus syndrome, cavernous sinus fistula syndrome. Foix syndrome, cavernous sinus thrombosis.	Unilateral.	Facial and ocular.	Rare.		Can be due to thrombosis, tumors, or trauma (75%) in the young, but is often due to an aneurysm in the elderly. Mucormycosis can cause rapid development of a cavernous sinus thrombosis when the fungi spread from the nose to the sinus.	With cavernous sinus thrombosis most have fever and they need large doses of antibiotics.	A cavernous sinus fistula causes chemosis, conjunctival congestion, lid swelling, pulsating exophthalmos, diplopia and reduced vision due to corneal exposure, secondary glaucoma and ischemia. Thrombosis of the cavernous sinus can cause paralysis of CNIII, IV, V, and VI as well as periorbital edema, bilateral proptosis, and chemosis.

Table 9

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with diabetic neuropathy. (Many have hypertension, kidney disease, atherosclerosis and arteriolarsclerosis.)	About 50% have some head pain. Many do not know they are diabetic. Diabetes can cause painful ophthalmoplegia.	Forehead or retrobulbar area, i.e. area served by the first division of the fifth cranial nerve. Ischemia may affect cranial nerves III, V or VI.		Chronic.	Diabetes mellitus produces occlusive disease of small vessels. The result is impaired circulation and the formation of small aneurysms and hemorrhages.	Insulin or oral antidiabetic drugs to control the diabetes. Cyclosporin is being tried for type I diabetes. Propranolol may help prevent the pain as well as promote insulin release.	The three classical manifestations are polydipsia, polyuria and polyphagia. Some have paralyses of CNIII, with pupil sparing but nearly 50% show pupillary involvement. Sudden paralysis of CN VI may occur but usually recovers. Children have subnormal accommodation as an early sign. The typical fundus signs include aneurysms, hemorrhages, and exudates.
Wallenberg's syndrome, posterior inferior cerebellar artery syndrome. (See acute cerebellar hematomas.)	Severe attack of vertigo causing the patient to fall.	Ipsilateral loss of pain and temperature sensation in face and throat and contralateral loss of pain and temperature sensation in trunk and extremeties.			Vascular disease of the brain stem, occlusion of the posterior inferior cerebellar artery.		Appears in the fifth decade. Ataxia, muscular hypotonia, vertigo, nausea, trouble swallowing, trouble speaking. Ipsilateral Horner syndrome, nystagmus, diplopia, ptosis.
Headache with intracranial tumors, abscesses, hematomas, shunts, angiomas, aneurysms. (Among those with cerebral aneurysms as many as 25% have migraine too.) (Fewer than 1% of patients with a headache have a brain tumor. Up to 40% of those with a brain tumor have no headache. Ten percent of patients with brain tumors first seek advice concerning their eyes.) (See sub-arachnoid hemorrhage, Bruns's syndrome, headache due to increased intracranial pressure and the headache with various tumors.)	Deep, dull, steady ache, mild to severe. Severity often changes when head position is altered. Severity is not a reliable guide to seriousness. Those with an organic problem may move slowly, walk with legs wide apart, do not swing their arms, and tend to have speech difficulties. Unruptured intra-cranial aneurysms are usually asymptomatic. Rupture of an intracranial aneurysm produces an intense headache.	Localized or generalized. A unilateral frontal or orbital headache with paralysis of CNIII can be caused by an aneurysm of the intradural portion of the carotid artery or the posterior communicating artery. Headache is localized over or near the site of the tumor in 33% of cases. The function of CNIII or CNV is frequently affected. (Subacute bacterial endocarditis is mostly due to streptococci or staphylococci and causes fever, headache, heart murmurs and hemorrhages in conjunctiva and retina, Roth spots.)	Intermittent or more or less constant. Type, location and frequency of headache depend on the location of the abscess, aneurysm or tumor. Often worst early in the day. Headaches are the initial symptom in 30% of patients with intracranial tumors. About 1 in 300 patients die of brain tumor.	Occur every day but vary in intensity. Beware if headache is of recent onset and is increasing in severity. An aneurysm of the internal carotid can cause an intermittent headache for years. The pain is ipsilateral and comes in episodes each lasting about one day.	Tumors and aneurysms cause headache by traction on or displacement of intracranial structures not by the increase in intracranial pressure. Rarely an aneurysm near the junction of the posterior cerebral and superior cerebellar arteries can cause paralysis of CNIII and usually causes some hemiparesis. The associated headache is not referred to the ocular region. Those with an intracranial abscess often have a history of infection, ear disease, sinusitis, lung abscess.	Treatment of the underlying condition. May need codeine or morphine for the pain but often ASA is adequate. Worsened by coughing, straining, exercise, defecating, sexual intercourse, weight lifting or by a sudden change in head position. Among those with a cerebral aneurysm a bruit may be heard. (About 15% of patients with multiple sclerosis have headache as a complaint in the initial bout of this illness.)	The photopsias, if any, pulsate more slowly than those of migraine. Slowly progressive weakness on one side of the body. Convulsions, tinnitus, ataxia, loss of balance, mental changes, visual disturbances, nystagmus, field losses, papilledema, and no visible venous pulse on the disc. With an aneurysm, the pupil on the affected side is almost always dilated and fixed. With paralysis of CNIII the affected eye turns down and out and has ptosis and mydriasis.

Table 10

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Tumors affecting the chiasma. Pituitary adenomas erode the sella in 80% of cases. Can be intrasellar or suprasellar.	Some but not all have headache. Headache occurs in 20% of patients with a pituitary adenomas.	Cranio-pharyngiomas may be suprasellar or intrasellar. Meningiomas may be tubeculum sellae (suprasellar), olfactory groove (presellar), or sphenoidal ridge (parasellar).	The pituitary gland is in the middle cranial fossa.		Craniopharyngiomas occur here. A tumor may erode into the paranasal sinuses and cause acute headache with oculomotor paralyses.		Pituitary adenomas cause bitemporal hemianopia and also impair the vision in 90% of these patients. A few suffer unilateral blindness and contralateral hemianopia.

Frontal lobe tumors are supratentorial.	These tumors frequently remain silent for a long time but eventually raise the intracranial pressure and cause headache.	The frontal lobes are in the anterior cranial fossa. If the tumor presses downward it causes the Foster-Kennedy syndrome with papilledema in one eye and optic neuritis followed by optic atrophy in the other eye. The Foster-Kennedy syndrome is not pathognomomic.			Likely tumors include medullablastomas, meningiomas, astrocytomas, glioblastomas or metastases from tumors in lung or breast.	Abscesses or aneurysms are able to produce similar signs and symptoms.	In the later stages frontal lobe tumors cause contralateral hemiparesis, anosmia and personality changes. Mental symptoms are common with these tumors. About 50% have papilledema, 5 to 10% have irregular nystagmus and 30% have visual field losses.
Temporal lobe tumors are supratentorial. (See Raeder's paratrigeminal syndrome.)	Headache is not a prominent feature in most of these patients. Intracranial pressure becomes elevated.	Tumors in the dominant lobe interfere with speech. Tumors in the left lobe impair memory and the ability to write and calculate. Tumors in the right lobe affect visual memory and the ability to read and learn.	The temporal lobes are in the middle cranial fossa.		Most of these tumors are gliomas, some are meningiomas, and only a few are angiomas, astrocytomas, medullablastomas or metastases from lung or breast.	With temporal lobe tumors patients may see organized visual hallucinations, objects, or persons instead of the visual aura associated with classical migraine.	Seizures are the initial sign in 40%. Look for exophthalmos, extraocular muscle paralyses, diplopia, ptosis, ipsilateral mydriasis with a fixed pupil and photophobia. Many have an incongruous, contralateral, upper-quadrant hemianopia and usually papilledema.
Parietal lobe tumors are supratentorial.	Focal tumors in this region cause visuospatial disorientation with a tendency to ignore the opposite side of the body. Intracranial pressure is elevated.	The parietal lobes are the upper central lobes of the cerebral hemispheres.			Probable tumors include medullablastomas, meningiomas (30%), astrocytomas, gliomas or metastases from lung or breast. If the tumor is anterior it causes focal sensory seizures. If the tumor is posterior it causes dyslexia, visual agnosia and primitive hallucinations.		Asymmetric optokinetic nystagmus is not uncommon. If the trigeminal nerve is affected, look for reduced corneal sensitivity and facial paralysis on the side opposite from the tumor and this combination is pathognomomic. Papilledema is rare but homonymous hemianopia is common.

Table 11

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Occipital lobe tumors are supratentorial. They are uncommon, about 25% are gliomas and 25% meningiomas.	Headache is not a prominent feature.	The occipital lobes constitute the posterior portion of the cerebral hemispheres and contain the visual cortex.	Tumors here are uncommon but if present cause epilepsy, mild mental disturbances and sometimes alexia, agraphia, micropsia, metamorphopsia and agnosia.		Tumors here may also be medullablastomas, astrocytomas, hemangioblastomas or metastases from lung or breast. Lesions affecting the supranuclear pathway cause loss of involuntary conjugate eye movements.		Seizures. Papilledema is usual and often extreme. About 85% have contralateral, homonymous, hemianopsia with macular sparing but sometimes the macula is also affected.
Supratentorial tumors can also develop from the corpus callosum, the pineal body or the midbrain region.	Midbrain tumors cause increased intracranial pressure.	When the cerebral peduncle and third nerve nucleus are affected there is paralysis of CNIII and contralateral facial palsy, Weber's syndrome.	When the red nucleus is affected there can be paralysis of CNIII with tremor and jerky movements of the contralateral side of the body, Benedikt's syndrome.	Patients with Nothnagel's syndrome show paralysis of CNIII, ataxia and gaze palsies. The lesion affects the superior cerebellar peduncle, red nucleus and fibers of CNIII.	Pineal tumors can cause ataxia, deafness, limitation of upward gaze, papilledema, transient amaurosis.		Parinaud's syndrome, can result from a midbrain tumor. They have paralysis of conjugate upward gaze as well as vertigo, ataxia and marked papilledema. The lids may show ptosis or be retracted. Pupils are dilated. These tumors produce disturbances of ocular motility but have no direct effect on the visual tracts.
Cerebellar tumors are infratentorial. (See also cerebellar hematomas.)	These patients often have a bifrontal headache and elevated intracranial pressure.	Pain in orbital, midfrontal, and neck regions. Among patients under 15 years of age 65% of all brain tumors are in the cerebellum.	The cerebellum is in the posterior cranial fossa.		Tumors can displace the cerebellar tonsils through the foramen magnum and cause impingement on the dorsal roots of the cervical nerves.	Surgery and radiation are required.	Classical cerebellar signs are ataxia, loss of balance, hypotonia and nystagmus. Seizures are common, nausea and facial paralysis often occur. With elevated intracranial pressure patients show weakness of conjugate gaze, nystagmus and papilledema with no pulse visible on the disc.

Cerebellopontine angle tumors are infratentorial.	Intermittent headache and raised intracranial pressure. These tumors may occur with neurofibromatosis, von Recklinghausen's syndrome.	Behind the ear. When an acoustic neurinoma develops on CNVIII it causes similar manifestations and can cause paralysis of CNVI. Contralateral hemiplegia is usual.		Chronic.	Cranial nerve pareses are to be expected. Facial pareses (CNVII). Tinnitus, deafness, and vertigo (CNVIII). Corneal anesthesia, and loss of sneeze reflex (CNV). Dysphagia (CNX).	Vascular lesions can produce similar effects.	Hypotonia, nausea, ataxia, contralateral hemiplegia, facial palsy, and vestibular nystagmus. Paralysis of the lateral rectus, corneal anesthesia, dry eyes and papilledema.
Infratentorial tumors in the pons and medulla.	Headache and raised intracranial pressure, but rarely papilledema.	Tumors of the medulla tend to cause early death so eye signs are infrequently reported.	The pons and the medulla are in the posterior cranial fossa.		Gliomas are the most common tumors here. They are likely to affect the nuclei of the cranial nerves and produce paralysis of CNV, VI, VII or VIII.	Early signs are cardiac irregularities, vertigo and trouble swallowing. The gaze centres are located in the pons.	Vestibular nystagmus is often present. Papilledema is uncommon, but can develop along with paralysis of the tongue in the late stages of medullary tumors. Pontine tumors rarely cause papilledema.

Table 12

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Infratentorial tumors in the pons and medulla oblongata.	These patients develop headache with raised intracranial pressure but only a few show papilledema.	Tumors of the medulla tend to cause early death so eye signs are infrequently reported.	The pons and the medulla are in the posterior cranial fossa.		Gliomas are the most common tumors here. They are likely to affect the nuclei of the cranial nerves and produce paralysis of CNV, VI, VII, or VIII.	Early signs are cardiac irregularities, vertigo and trouble swallowing. The gaze centers are located in the pons.	Vestibular nystagmus is often present. Although papilledema is uncommon it can occur along with paralysis of the tongue in the late stages of medullary tumors. Pontine tumors rarely cause papilledema.
Headache with raised intracranial pressure. (Not all patients with elevated intracranial pressure have a headache.) (See intracranial tumors, Bruns' syndrome, hematomas, aneurysms and hemorrhages.)	Deep, dull, ache, may change when head position is changed. Tumors affecting the post chiasmal visual pathways generally cause increased intracranial pressure and headache. These tumors may be supratentorial or infratentorial.	Pseudotumor cerebri is another potential cause especially in young obese females. The cerebral edema and narrowed small ventricles and increased intracranial pressure cause papilledema.			Tumors, aneurysms, occlusions, hemorrhages and disturbances of the circulation of the cerebrospinal fluid. Cerebral edema occurs in many conditions. In infants the fontanelles will bulge. Consiousness may fluctuate.	Worsened by coughing, straining, exercise, sexual intercourse, defecating, weight lifting.	May suffer blackouts, projectile vomiting without nausea, anorexia, convulsions, ataxia, loss of balance, circulatory and respiratory changes, psychic disturbances. Some show reduced corneal sensitivity, disturbed function of extraocular muscles, nystagmus, field losses, papilledema, and have no visible pulse on the disc. A few develop bilateral exophthalmos.
Bruns' syndrome, postural change syndrome. (See intracranial tumors.)	Severe headache but discomfort varies with head position.	Affects cranial nerves III, II and others.	Free of symptoms between attacks.	Paroxysmal attacks of vertigo, headache and projectile vomiting on change of head posture.	Tumors or other lesions of third, fourth, or lateral ventricles which obstruct flow of cerebrospinal fluid. Some are due to cysticercus infection.	Signs and symptoms can be altered by changing head position. Partial ophthalmoplegia can be precipitated by postural changes.	Ataxia, nausea, vomiting, vertigo, tachycardia and syncope. Paralysis of CNIII often occurs. Transient blindness, flashes of light, papilledema.
Headache with an acute cerebellar hematoma. (Cerebellar hematomas constitute 10% of all hemorrhages into brain tissues.) (See cerebellar tumors and the Wallenberg syndrome.)	Acute pain in the head.	Occipital.	Sudden onset.		Hemorrhage into the cerebellum. Traction on or displacement of pain sensitive structures. The posterior inferior cerebellar artery has been called the artery of thrombosis. It is a branch of the vertebral artery.	Prompt treatment, surgical drainage. A cerebellar hematoma is life threatening.	Many have hypertension, ataxia, brain stress signs, nausea, vomiting. Look for papilledema.

Table 13

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Terson's syndrome, multiple cerebral aneurysms. Subarachnoid hemorrhage syndrome. (At least 40% die if they have a major bleeding episode.) (See stroke headache, ischemic cerebrovascular disease, and effort headache.)	Severe, throbbing pain in the head. Sudden and severe supraorbital pain. Headache is often the chief symptom but 10% suffer seizures too. A congenital cerebral aneurysm is usually asymptomatic until it ruptures, this can occur in a migraine attack or following trauma.	Sometimes occipital but often begins above one eye. Pain in the back and legs develops later. An aneurysm of the supraclinoid, internal carotid, or posterior communicating arteries causes paralysis of extraocular muscles and pain in the area supplied by the ipsilateral trigeminal nerve.	Constant. Aneurysms of the posterior communicating artery can cause head pain before the subarachnoid hemorrhage occurs. Trauma can precipitate the hemorrhage.	Sudden onset and severe for about a week; all gone in two months providing the victim survives. Recurrent hemorrhages, if they occur, are likely to cause death.	Traction on pain-sensitive structures produces the headache. Sub-arachnoid hemorrhage is often due to rupture of an aneurysm. The aneurysm is usually located at the junction of the internal carotid and posterior communicating arteries. The stroke of the young patient. Death can occur rapidly.	Surgical drainage but usually not treatable in the acute stage of a head injury. Diagnosis of subarachnoid hemorrhage is aided by spinal puncture.	Symptoms of arteriovenous malformations often resemble those of migraine but the aura flickers more slowly than in migraine. Some become drowsy or comatose. Fever, stiff neck, bilateral partial or complete paralysis of CNIII, ptosis, vitreous hemorrhages, ipsilateral mydriasis and photophobia. Papilledema if it occurs is a late sign.
Headache with subdural hematomas. (Subdural hemorrhages are more common than extradural hemorrhages.)	Most have a severe headache which develops rapidly. All the signs tend to fluctuate. Loss of consciousness occurs in the acute type.	Pain at first is on the side of the injury but later all over the head.	More apt to occur in chronic alcoholics.	Acute type or chronic type can follow head injury. Pain tends to become constant.	Trauma affecting the middle meningeal artery. Bleeding may be from a vein close to the sphenoidal ridge. Hematomas can occur spontaneously and produce chronic headache.	Surgical drainage. Signs may not appear until weeks after the trauma. Prognosis is poor for infants and better for adults.	Chronic type causes: dizziness, drowsiness, seizures, confusion, hemiplegia, diplopia, fixed dilated pupil on the side of the lesion (Hutchinson's pupil), papilledema, field defects, and vestibular nystagmus.
Headache with extradural hemorrhages.	First become unconsious then have a lucid interval of minutes to hours, followed by headache, nausea and vomiting.				Follow injury to the anterior branch of the middle meningeal artery; often the result of a linear fracture of the skull.	Surgery is required.	This is an emergency. Patients usually enter a coma, and can die promptly.
Headache with meningeal irritation, chronic meningitis.	Headache is dull to severe, gradual onset. More rapid onset if due to intracranial bleeding. Increased intracranial pressure.	Bilateral or generalized or over the vertex and radiating down the neck. Cranial nerves V, VII, IX and X and the upper cervical nerves are sensitive to pain.	More or less constant. Child is much more uncomfortable if moved.	Until the inflammation subsides.	Meningeal infections including those associated with syphilis, tuberculosis, meningococcal or other infections including viral.	Treatment of the underlying condition. May need codeine or morphine to ease the pain and i.v. antibiotics to control the infection.	Likely to be acutely ill with fever, nausea, vomiting, cranial nerve palsies, delirium, lethargy and a stiff neck. Photophobia and papilledema.

Table 14

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Ramsay-Hunt syndrome, geniculate neuralgia, otic neuralgia, Hunt's syndrome, herpes zoster oticus. (See Melkersson-Rosenthal syndrome.)	Severe neuralgic pain. The geniculate ganglion is the sensory ganglion of CNVII. (Similar acute and prolonged local pain usually precedes herpes zoster vesicles on the skin.)	Deep pain in ear. Cranial nerve VII. Herpes viruses may lodge in the geniculate ganglion or in peripheral ganglia of CNIII, IX or X.	Paroxysms of pain occur in the ear when the geniculate ganglion, and the nervus intermedius (Wrisberg) of the seventh nerve are involved.	Pain precedes vesicle formation by several days and may persist after the lesions have disappeared, postherpetic neuralgia.	Herpetic infection of the geniculate ganglion. Herpes zoster (HSV-3) causing an eruption on the tympanic membrane, the external auditory meatus and the concha of the auricle.	Corticosteroids, antidepressants, lithium and phenothiazines have all given help.	Facial paralysis. Hearing may be affected; tinnitus and sometimes vertigo and depression. Dry eyes, dry throat, reduced corneal sensitivity, risk of corneal ulceration, nystagmus.
Melkersson-Rosenthal syndrome, geniculate ganglion syndrome, facioplegic migraine. (See the Ramsay-Hunt syndrome, tic douloureux and Quincke's edema.)	This form of facial palsy is often associated with a migraine type headache.	Face. Facial palsy may be unilateral or bilateral. Cranial nerve VII and its geniculate ganglion are affected.	Periodic.	Chronic. Begins in childhood or early adult life.	Cause obscure but often inherited as an autosomal dominant trait. Possibly the result of a viral infection damaging the parasympathetic cells or fibers in the geniculate ganglion.	Corticosteroids and analgesics are used to treat this type of angioneurotic edema.	Recurrent or chronic painful facial edema, and paralysis; swelling of the lips; congenitally fissured tongue. Some have lagophthalmos, increased lacrimation, blepharochalasis, corneal ulcers, corneal opacities, retrobulbar neuritis and exophthalmos.

Tic douloureux, gasserian neuralgia, trigeminal neuralgia, Fothergill's neuralgia, trifacial neuralgia. One type is called supraorbital neuralgia. (See Raeder's syndrome and Rosen's neuralgia.)	Sharp, stabbing, high intensity pain and burning sensation with spasms of facial muscles. The gasserian (trigeminal or semilunar) ganglion is the sensory ganglion of the trigeminal nerve CNV.	Pain in the face in the eye region, slightly more often on the right side. Pain is limited to one side of the face in the area supplied by the second and less commonly that supplied by the first and third divisions of CNV.	Episodic. Worst in March and October. Pain episodes recur for two or three months at a time. Pain is more constant in the rare cases where a tumor is responsible.	Brief 20 second stabs of pain, repeated for up to an hour. Condition may recur for years but usually remits spontaneously.	Cause may be a blood vessel impinging on a branch of the fifth nerve. Precipitated by stimulation of a trigger area 3 or 4 mm in diameter near the nose or mouth. May accompany multiple sclerosis or follow a herpes infection. Be sure no tumor is present.	Anticonvulsant drugs help but the rationale is not clear, e.g. diphenylhydantoin sodium (Dilantin) 100 mg b.i.d. or t.i.d. or carbamazepine 200 mg b.i.d. or t.i.d. will control the pain in 66% of cases. Some use baclofen, amitryptyline, diazepam, or surgery including radiofrequency heating.	Rare, (estimated to affect 15 people in 100,000) slightly more common in men. Usually affects those over 50 years of age. Most have a trigger area. Generally no nausea, no vomiting, no scotomata. Rarely increased lacrimation and ipsilateral conjunctival hyperemia. The neuro-paralytic keratitis can lead to corneal ulceration or perforation.
Glosspharyngeal neuralgia. (See tic douloureux, Sluder's syndrome, Raeder's syndrome, Costen's syndrome, Charlin's syndrome, and tympanic plexus neuralgia.)	Sharp, stabbing, severe, unilateral, neuralgic pain. The superior and inferior (petrosal) glossopharyngeal ganglia contain cell bodies of the afferent fibers of the glossopharyngeal nerve.	Begins in the back of the throat and radiates to the angle of the jaw, the tongue and the neck and extends to the ear. Cranial nerve IX.		Paroxysmal episodes come in groups.	Obscure. Possibly viral. Some have a trigger area in the tonsillar region.	Treatment is similar to that of trigeminal neuralgia, tic douloureux. Is not a self limited condition. May require surgery of the glossopharyngeal nerve.	Affects about one person in a million. Most of these patients are over 50 years of age. Some have cardiac slowing which can lead to syncope.

Table 15

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Tympanic plexus neuralgia, Reichert's syndrome. (Compare with Rosen's neuralgia). (See glossopharyngeal neuralgia.)	Sharp neuralgic pain. The chorda tympani nerve originates from the facial nerve and carries parasympathetic and sensory fibers.	In the vicinity of the external auditory meatus but spreads to the face and tongue and behind the ears.		Paroxysmal.	Possibly viral. Involvement of the tympanic branch of the glossopharyngeal nerve, cranial nerve IX.	Treated by sectioning the nervus intermedius, (a part of CNVII), the chorda tympani or CNIX.	Rare. (Rosen's neuralgia is tic douloureux of the chorda tympani.)
Ménière's syndrome, Ménière's disease, vestibular migraine, eighth nerve neuralgia. (Lermoyez's syndrome is similar but in the Lermoyez syndrome hearing improves during the vestibular attack.) (See Cogan's syndrome and basilar artery migraine.)	Headache varies in severity as do the vertigo and tinnitus. Patient may fall down when the attack begins. Ménière's syndrome may be confused with labyrinthitis.	Abrupt onset of an occipital headache (on the side of the tinnitus) or a tight sensation in the back of the head and neck. Cranial nerve VIII. Deafness may be unilateral or rarely bilateral and is not total.	Typically occurs in episodes appearing every 4 or 5 weeks and separated by symptom-free periods of months or years.	Each episode lasts up to one hour but more than one attack can occur in a single day.	Disorder of the endolymphatic system of the inner ear. Paroxysmal labyrinthine hydrops. Neuralgia of CNVIII. Distended arteries in the posterior fossa impinge on cranial nerves V, VII, VIII, IX and X. Local autonomic dysfunction.	Diazepam i.v., dimenhydrinate orally or i.m., meclizine orally or i.m., cyclizine orally or i.m. or scopolamine transdermally.	Most apt to appear in males in their fifties. Vertigo, tinnitus, nausea, some permanent hearing loss. Room seems to revolve even when the eyes are open. Cheek on the affected side feels dead. Nystagmus with the fast component toward the normal ear. Rarely diplopia and corneal anesthesia.
Sluder's syndrome, Sluder's lower-half headache, sphenopalatine ganglion neuralgia, atypical facial neuralgia, (See cluster headaches. Both may be called sphenopalatine ganglion neuralgia.) (See Charlin's syndrome and vidian neuralgia.)	Continuous pain, burning, deep-seated, nagging and fairly severe. The sphenopalatine (pterygopalatine) ganglion is a parasympathetic ganglion in the pterygoid fossa and receives preganglionic fibers from the facial nerve CNVII.	Usually unilateral or rarely bilateral, in eye, nose, orbit, temple and upper jaw. Pain radiates from the lower face e.g. palate up to the ear. Some have pain in the mastoid region. Rarely pain extends to the occiput. Cranial nerves III, V or VII can be affected.	Frequent episodes; freedom from pain between attacks. Attacks do not appear in groups as they do in cluster headaches, glossopharyngeal neuralgia, tic douloureux or vidian neuralgia.	Episodes last for minutes to days. Condition may persist for years. Residual tenderness of the neck muscles tends to persist.	Probably due to vasodilation of branches of the external carotid, possibly the sphenopalatine branch of the internal maxillary artery. Some blame intranasal or sinus pathology or viral infections. Worsened or precipitated by alcohol, tobacco, or a change in climate. Hysterical mechanisms seem to be involved.	Ergotamine may be helpful but typically treatment is difficult. Beware overuse of analgesics. Correct refractive errors and ocular muscle imbalance.	More women than men. Younger patients. Not a well-defined syndrome, may be a group of similar conditions. Tender spot 3 to 5 cm behind the mastoid but no trigger area. No Horner's syndrome, no gastrointestinal upset. There is a vascular type with unilateral, throbbing, midfacial pain in which facial flushing, rhinorrhea and excessive lacrimation occur.
Vidian neuralgia, Vail's syndrome. (See Sluder's syndrome.)	Fairly severe neuralgic type pain. The vidian nerve is formed by the union of the great superficial petrosal and the deep petrosal nerves.	Usually unilateral. Pain in nose, face, eye, ear, head, neck and shoulders.	Pain appears in acute attacks often during the night.	Episodic.	The vidian nerve passes close to the sphenoid sinus on its way to the sphenopalatine ganglion. Irritation or inflammation of the vidian nerve may be due to a viral infection.		

Table 16

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Raeder's paratrigeminal syndrome, the paratrigeminal neuralgia syndrome. A similar symptom complex has been called Symond's type of migraine. (See Horner's syndrome, tic douloureux, cluster headaches, and glossopharyngeal neuralgia.)	Headache is severe, neuralgic or throbbing.	Unilateral, fronto-orbital headache and ipsilateral periocular pain. Facial pain in the region supplied by the first division of the trigeminal nerve CNV. Affects not only the sympathetic fibers but sometimes CNII, III, IV, VI and any division of V.	Recurrent morning headaches. Headache often wakens the patient from sleep. Some have a cluster of headaches, others have a single episode lasting hours to weeks. These two types are benign.	Generally clears spontaneously in a few weeks. Transitory but prognosis is guarded. These headaches are ominous if any other neurological signs develop.	Obscure, possibly meningitis or an aneurysm of the internal carotid affecting CNV and its sympathetic fibers. Some are due to a neoplasm in the middle cranial fossa. A few have fibromuscular dysplasia along the carotid artery. Many have high blood pressure.	Check for a possible neoplasm. Treat the high blood pressure if it is present. Some obtain relief of the headache by oral prednisone.	Mostly middle-aged males. Weakness of jaw muscles, nausea, vomiting but no aura. Suddenly develop an incomplete Horner's syndrome, (ptosis and miosis) but the sweating mechanism is preserved. Severe ocular pain, excessive lacrimation, decreased corneal sensitivity, rarely diplopia and scotomas.
Charlin's syndrome, the nasociliary syndrome, migrainous neuralgia, Harris' neuralgia. (See cluster headache. Both may be called ciliary neuralgia). (See Sluder's syndrome.)	Severe neuralgic pain. The ciliary ganglion lies deep in the orbit and is a parasympathetic ganglion receiving preganglionic fibers from the oculomotor nerve CNIII.	Unilateral in eye, orbit, temple, cheek, jaw and at the root of the nose. Sharp pain at the inner angle of the eye where the nasociliary nerve exits on the side of the nose. CNV.	Long intervals between attacks.	Paroxysms of pain last for minutes to hours.	Neuritis of the nasociliary nerve or of the ciliary ganglion. A virus could cause this type of ciliary neuralgia.	Anesthetic applied to the nerve in the anterior portion of the nasal fossa gives temporary relief.	More common in men. Inflammation of the anterior segment of the eye on the ipsilateral side. Excessive lacrimation, rhinorrhea, blepharospasm, superficial keratitis, photophobia, iritis.
Headache with Bell's palsy, idiopathic facial paralysis.	Local ache. The petrosal and the stylomastoid arteries supply the nerve in the Fallopian canal.	Pain in ear and mastoid; tingling or numbness in cheek or mouth. Face feels stiff and is pulled to one side. Paralysis of CNVII.	Sudden onset in adults.	Generally they recover gradually. Pain eases after a few days. Full recovery in 80%.	Unknown, possibly viral; have inflammation and edema of the facial nerve. Edema in the Fallopian (facial) canal or in the stylomastoid foramen.	Corticosteroids but some studies show that these drugs provide little help. Electrical stimulation often helps restore facial muscle function.	A few have permanent facial paralysis. Hyperacusia, changes in ability to taste, corneal ulcer, ectropion, lagophthalmos, nystagmus, epiphora, ocular irritation.
Horner's syndrome, Bernard-Horner syndrome. (Forms part of many other syndromes.) (Often occurs with cluster headaches and Raeder's syndrome.) (See the Barré-Liéou syndrome.)	Usually no pain but have an oculosympathetic paralysis. Horner's syndrome with heterochromia iridum is called Passow's syndrome.				Impairment of the sympathetic supply to the eye can follow damage to any of the three neurons in the chain. Can be due to birth injury, follow thyroid surgery, or be due to other causes including bronchogenic carcinoma.	Identifying the site of the lesion and treating accordingly. If developing in a middle aged smoker check for cancer of the upper lobe of the lung.	Ptosis, miosis and ipsilateral anhydrosis. If due to birth trauma the iris will be pale in the eye on the affected side. Heterochromia iridum. Rarely an affected eye develops an abnormally shaped pupil.

Table 17

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Tolosa-Hunt syndrome, Hunt's syndrome, superior orbital fissure syndrome, painful ophthalmoplegia syndrome. (See complicated migraine and the carotid artery syndrome.)	Pain can be severe and is steady. In the syndrome of the apex of the orbit, (Rollet's syndrome) CNII is also involved.	Unilateral, retro-orbital pain in the area supplied by the first division of CNV. Followed by paralysis of cranial nerves III, IV, or VI.	Attacks recur at intervals of months or years.	Symptoms persist for days or weeks. Disease is usually self-limited.	A non-specific granulomatous inflammation involving any or all of the nerves traversing the superior orbital fissure or cavernous sinus. Trauma, aneurysms or neoplasms are potential causes.	Systemic steroids may be needed for months. Check for diabetes, carotid aneurysms, or tumors. Have an elevated erythrocyte sedimentation rate.	Those affected are usually over 40 years old. Mild exophthalmos. The painful unilateral ophthalmoplegia recovers slowly. Ptosis, sluggish pupillary reactions (in 2%), scintillating scotomas; corneal sensitivity is reduced.
Guillain-Barré syndrome and its variants such as the Miller-Fisher syndrome and Landry's acute ascending paralysis.	Pain or paresthesia precede the flaccid paralysis. Paralysis of any cranial nerve especially VII, IX, or X.	Weakness, pain and progressive paralysis in legs. Some have areas of relative anesthesia. Loss of tendon reflexes.			Acute polyradiculoneuronitis with facial paralysis following an infection. Excess protein in cerebrospinal fluid.	Usually make a complete recovery within a few months but a few die from respiratory paralysis.	Paroxysmal hypertension, ataxia, weakness. Sometimes fixed dilated pupils, ophthalmoplegia, lagophthalmos, optic neuritis, papilledema, optic atrophy.

Cogan's syndrome, Cogan's non-syphilitic interstitial keratitis syndrome. (See Ménière's syndrome.)	Abrupt onset of ocular pain or vertigo and tinnitus with hearing impairment.	Earache and some pain in the eye. Ocular or aural symptoms may be first to appear. Cranial nerve VIII.	This is a rare condition which usually results in some permanent damage to both hearing and labyrinthine function.	Begins on the average at 29 years of age, and persists for a few months.	Considered to be an immune vasculitis. Risk of vascular occlusions. May be related to periarteritis nodosa.	Topical corticosteroids e.g. dexamethasone phosphate (0.1%) one drop every 2 hours for 7 to 14 days. To prevent profound deafness requires systemic administration of prednisone 80 mg a day.	Mostly young adults. Vestibular and auditory dysfunction with vertigo and tinnitus. Some hearing reduction is permanent. Bilateral, subepithelial interstitial keratitis and stromal edema. Ciliary hyperemia; photophobia, blepharospasm, episcleritis, nystagmus. Transient reduction in visual acuity.
Effort or exertional headache; cough headache; post-coital headache; benign orgasmic cephalalgia. (See Terson's syndrome.)	Throbbing, severe. Heart rate and blood pressure increase during intercourse. (Similar headaches can accompany the Valsalva maneuver, forced expiratory effort against a closed airway.)	Usually frontal, behind the eyes but some have an occipital ache.	Occurs in some individuals with orgasm, or immediately after similar exertion. In eyes with controlled narrow-angle glaucoma sexual intercourse in the prone position can precipitate an attack of glaucoma.	Usually brief, up to an hour or so but sometimes does not develop until 2 hours later. A cough is likely to aggravate any headache especially one with brain tumor or elevated intracranial pressure or sinusitis.	Exertional or effort headache can be precipitated by weight lifting or by running or by sexual intercourse. Some have a malignant type associated with a tear in subarachnoid membranes or an actual hemorrhage.	Ergotamine tartrate or methysergide prophylactically is indicated for effort headache. Subarachnoid hemorrhage can be diagnosed by spinal puncture.	Men are more often affected. Effort, or straining raise the blood pressure but this particular headache is not dependent on having chronic high blood pressure and has no associated neurological signs or symptoms unless there is a subarachnoid hemorrhage.

Table 18

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with Paget's disease, osteitis deformans. (There is also a condition of congenital hyper-phosphatasemia, juvenile Paget's disease.)	Headache is mild, burning and only rarely severe, however many of those with Paget's disease have no headache.	Localized or generalized. Tender areas on scalp. Eventually pain in back and limbs.	Intermittent or constant.	Prolonged. (The hereditary autosomal recessive type has its onset before 4 years of age. The affected child suffers, dwarfism, osteoporosis, skull thickening, kyphoscoliosis, bowed legs, and has angioid streaks in the fundus, optic atrophy, macular changes and blue scleras.)	Cause of the adult type is unknown. Rate of bone resorption and formation both increased in the spinal column and skull; the result is compression of the brain and the cranial nerves. Many die of cardiovascular disease, some have hypertension, and bleeding in their gastrointestinal tract.	Analgesics and sedatives offer some help for the headache. Calcitonin or mithramycin help control Paget's disease. Patients have high serum levels of alkaline and acid phosphatase.	Mostly males over 40 years of age. Skull increases in size, becomes tender and thickened, but bones become more porous. Skin pigmentation increases. EOM palsies, proptosis, optic atrophy and in 10% angioid streaks. Paget's disease with angioid streaks is called Terry's syndrome.
Post-traumatic, or post-concussion headache, occipital neuralgia. Craniocervical or punch-drunk syndrome. Friedmann's syndrome is traumatic encephalitis. (Kummell's disease is due to traumatic damage to the vertebrae.) (Cervical disc disease or injury often follows an accident.) (A post-traumatic headache occurs in at least 33% of all who are hospitalized for head injury.) (See depression headaches, and the Barré-Liéou syndrome.)	Variable intensity. Type 1 have a steady pressure sensation or band-like ache. Type 2 have a type 1 ache plus a local tender zone. Type 3 have a throbbing, unilateral ache in attacks.	Localized to the site of the injury or generalized or in the forehead and temples. Back and neck pains are often associated. Type 3 is often unilateral. Affects CNV and CNVIII. (Lesions of the cervical spine cause the Barré-Liéou syndrome also called the posterior cervical sympathetic syndrome.)	Variable frequency. Can change with barometric pressure. Type 1. Intermittent but recurs for years. Type 2 continuous or intermittent. Type 3 recurs in short spasms.	Signs and symptoms usually develop within two days of the trauma but in a few patients appear weeks later. Variable duration. Most such headaches fade in two months. Some complain of discomfort until all legal actions are settled. Even years later a few continue to have headaches and dizziness. Whip-lash type injuries often produce prolonged discomfort.	Even minor injury can cause this headache. Hyperextension or hyperflexion of the cervical spine. Type 1 is related to muscle contraction. Type 2 to local tissue damage including scarring, fibrosis, adhesions, disc lesions, hemorrhages. Small focal hemorrhages in the cerebrum and/or cerebellum. Type 3 is related to dilation of branches of the external carotid, a vascular mechanism. Psychological factors are important.	Time, massage, warmth, analgesics, propranolol, antidepressants. Phenobarbital provides relatively short periods of relief. Worsened by some head positions, by drinking alcohol, by emotional upsets or by changes in barometric pressure. Type 3 can be helped by cold compresses and by ergotamine. All need patient understanding and often psychotherapy as the depression tends to persist.	Many become tense, anxious, irritable, dizzy, photophobic, and unable to do prolonged reading. Vestibular (CNVIII) dysfunction occurs in over 50% of cases. Personality changes, memory impairment, resentment and emotional reactions. Patients with type 3 often have anxiety, depression and sometimes nausea. Look for vertical nystagmus, impaired convergence, reduced amplitude of accommodation and sometimes Horner's syndrome, but in type 3 the pupils are dilated, not miotic.
Lumbar puncture headache, leakage headache, post-spinal-tap headache, low pressure headache.	Severe or rarely mild headache. Dull, deep and can be throbbing type.	All over the head, especially frontal or occipital and neck regions. Often extends into a backache.	Constant. Begins within 24 hours of lumbar puncture in about 50% of these patients.	Twenty-four to 48 hours but longer if a spinal anesthetic or a contrast medium has been injected into the spinal column.	Follows withdrawal of 20 ml or more of spinal fluid. Leakage of spinal fluid allows traction or displacement of intracranial structures. Muscle contraction also occurs. Strong osmotic agents can produce a similar headache.	Recumbent position, no pillow. Hydration. Analgesics or ergotamine or DHE45. Use of a fine needle when performing the puncture and employing an oblique entry decrease the leakage of spinal fluid.	Lumbar puncture often causes dizziness, nausea and vomiting and sometimes blurred vision. A lumbar puncture is dangerous if the intracranial pressure is elevated. The cranial structures can herniate into the foramen magnum.

Table 19

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache with toxic or deficiency states, uremia, alcoholism, nephritis, and anemia. Carbon monoxide or other poisons, e.g. lead, arsenic, nitrites, morphine. Infections, mononucleosis, gastroenteritis or any condition causing fever. May accompany drug abuse or hangover or follow a seizure. (See dietary migraine.)	Moderate to severe, pulsating headache. A high fever in children can cause seizures and vomiting.	Generalized. Headaches with fever are dull and seem deeply situated. Headache and fever accompany subacute bacterial endocarditis. (See headache with intracranial tumors, abscesses etc.) Headache with meningitis can be severe.	Can develop at any age. Often daily. Constant. Following a seizure many develop a headache.	Depends on cause.	Fever and septicemia are the third most common causes of headache. Distention of cerebral and pial arteries is the principal mechanism. History of exposure to toxin. Can also be due to hypoglycemia, anemia, hypoxia, noxious fumes, hangover. The mechanism by which ice cream in the mouth causes a headache is not known, ischemia (?).	Controlling or eliminating the cause. Worsened by exercise or exertion or by lying down. Some people overdose themselves with analgesics so what they need is a reduction in their headache medications.	Headache usually accompanies a fever especially in children. Look for other signs caused by the noxious agent. Sodium nitrite is in hot-dogs or can be contacted in industrial situations. Monosodium glutamate (MSG) in many foods can cause a headache. The Chinese restaurant syndrome headache develops in susceptible individuals about 20 min. after the MSG compound is ingested.
Coffee hunger headache, caffeine withdrawal headache.	Definitely unpleasant.	Frontal.	Often on Sunday morning or other occasions when the usual intake of coffee is delayed.	Until coffee or caffeine containing soft drinks are consumed.	Is associated with dilatation of intracranial and apparently extracranial arteries as a result of caffeine withdrawal.	Coffee acts as a vasoconstrictor and frequently helps to relieve other types of headache.	Very common. An overdose of caffeine can cause a similar headache, with confusion, anxiety, tremors, vomiting and diarrhea.
Periodic headaches, also called cyclical headaches, menstrual headache. (See also migraine.)	Mild to moderate.	No specific location. Usually have abdominal discomfort in the form of cramps.	Once a month. Some women have a headache at both midcycle and just before or during menstruation.	Usually one or two days.	Related to ovulation-menstruation cycle. Possibly an estrogen-pituitary imbalance. Premenstrual tension. Premenstrual syndrome, PMS.	Mild analgesics. Some non-steroidal anti-inflammatory drugs are particularly helpful e.g. mefenamic acid or naproxen sodium for premenstrual headache.	The headache occurring at mid cycle, i.e. at ovulation time is called Mittelschmerz. Fluid retention usually occurs just before menstration.

Table 20

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Primary muscle contraction or anxiety-tension headache. Secondary muscle contraction headaches: spastic, myositic or myalgic headache, and cervical arthritis. (Stress, anxiety and emotional tension are the most common causes of headache. The resulting muscle tension causes at least 60% of all headaches.) (See psychogenic headache, post-traumatic headache, depression headache, vasomotor, rhinitis, combined headaches and Roy's ocular migraine.)	Feeling of a tight band around the head. Dull ache, gradual onset, not severe, only rarely pulsating. The acute type is mild and usually self-limited. The chronic type requires treatment. Usually no family history of a similar headache.	Usually bilateral but can be unilateral, vertex, fronto-occipital, temporal and generally extends into neck and shoulders. A real pain in the neck. Often have local tender areas in scalp, neck, or trapezius muscles.	Chronic recurring pain. Often occurs daily, especially toward the end of the day. Sometimes appears in the early hours of the morning but rarely awakens the patient. May increase in frequency at menopause.	Individual episodes last for hours or days but condition is prolonged for months or years. Condition may be acute or chronic. Trouble getting to sleep. Patient may become a hypochondriac.	Emotional factors, repressed hostility, stress, or let down from stress. Holding the head in an unusual position. Disorders of the cervical spine. Muscle tension and tension on the aponeurosis of the frontalis and occipital muscles causes scalp ischemia so the mechanism is vascular. Muscle contraction usually develops with any headache. Pain or stress can cause muscle tension and muscle tension can cause pain.	Patient needs to learn to understand the mechanism. Massage, warmth, gentle exercise, ASA, diazepam, propranolol or muscle relaxants. Helped by biofeedback techniques, by tricyclic antidepressants e.g. amitriptyline and by coffee. Worsened by ergotamine tartrate or by cold. Be sure no organic problem exists and that refractive errors and ocular muscle imbalances are properly corrected.	Often begins in patients between 20 and 40 years of age. Affects more men than women, rare in children. No prodromata, nausea or vomiting but may have anorexia. A common complaint is chronic fatigue. Some have a stiff neck. Stress probably plays a role in all types of headache. Depression often coexists and requires treatment. Overactivity of the sympathetic system causes dilated pupils and photophobia.
Barré-Liéou syndrome, cervical migraine, the posterior cervical sympathetic syndrome. (See the post-traumatic headache.)	Headache.	Occipital and pain in eyes and ears. Affects cranial nerves V and VIII.			Arthritis or trauma of the cervical spine irritate the sympathetic plexus around the vertebral arteries.		Vertigo, tinnitus, vasomotor disorders, facial spasm, stiff neck. Transient diplopia or loss of vision.
Psychogenic, emotional, conversion-hysteria headaches. (Also called tension headaches.) (About 50% of psychiatric patients report pain of some type.) (See combined headaches, depression headaches and post-traumatic headaches.)	Frequently the headache is constant, vise-like, hat-band or stabbing type. Unvarying intensity is suspicious. Headache descriptions tend to be vague or imaginatively detailed. They seek attention.	Bitemporal, vertex, or generalized. Reported distribution of pain does not follow anatomical features. May have a stiff neck.	Daily, constant. Unpredictable onset. Usually begins in third decade and affects more women than men.	Prolonged but in many cases the headache does not interfere with sleep or play. Often associated with a hypochondriacal state. Headache shows little or no change with the normal physiological changes such as puberty, pregnancy or menopause.	Insecurity, stress of daily living, anxiety, even headache from another cause can induce this kind of headache. Mechanism may be muscular, vascular or hormonal. Even if the cause is obscure the pain is real and not based on a conscious attempt to deceive. Depression is likely to develop.	Ruling out organic causes. May need more than elementary psychotherapy. Some need tranquilizers, antidepressants, or phenobarbital. They do not respond well to the usual analgesics. Avoid overuse of analgesics. These patients are reluctant to admit relief.	Suffer from anxiety but often appear outwardly calm and relaxed. Most have trouble getting to sleep. Emotional lability. Some become hypochondriacs. Usually a long history including complaints of fatigue, backache and anorexia. Bizarre symptoms are sometimes reported. Pupils may be large.

Table 21

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Depression headache. (Up to 90% of all headaches seen by internists and family practitioners are associated with mental depression.) (See post-traumatic headache, combined headaches and psychogenic headaches.)	Various types of headache; can be hatband type or dull, all around the head or a mild constricting type headache.	May be all over the head or more at the back of the neck or the base of the skull. A stiff neck is a common complaint.	Almost daily. Worse on weekends and holidays. Often occurs between 4:00 and 8:00 a.m. or p.m. Some anxiety is always present. These patients tend to awaken early and lie in bed worrying.	Onset at any age. Persists for weeks or months; rarely entirely free of pain.	May be caused by the presence of a headache due to some other cause. Can follow trauma. A deficiency of catecholamines in the brain has been reported.	Be certain no organic disorder exists. Antidepressants e.g. amitriptyline are often required. Eased by getting up and moving about. Correct refractive errors and ocular muscle imbalance after other care has been initiated.	Affects both sexes equally. Sometimes headache is the only complaint. Often appear outwardly calm and relaxed but report inability to concentrate, fatigue, loss of drive, feel sad, have poorer memory, and frequently have gastrointestinal problems.
Sinus headache. (Sinus infection is the commonest infective cause of headache, but actually causes less than 2% of headaches.) (See vasomotor rhinitis.)	Pain is dull, deep not throbbing. Children with Citelli's sinusitis syndrome have diffuse pain which may be referred to the occiput, mastoid or temples. Complain of loss of memory, inability to concentrate and somnolence.	Jaw, forehead or teeth. Fifth cranial nerve, 2nd division. Frontal but in chronic cases often becomes occipital. If the sphenoid sinus is involved the pain is deep in the head or at the vertex. If the ethmoid sinus is infected the pain is over the bridge of the nose or behind the eyes. Steady pain is felt over the infected sinus if the frontal or maxillary sinuses are involved.	Periodic or constant. May develop or change in association with changes in barometric pressure or humidity.	Worst in the morning except when the maxillary sinus is the offender, then it is worst at the end of the day. Pain is a prominent symptom with acute sinusitis but not with chronic sinusitis.	Inflammation of the turbinates, ostia and ducts. Obstruction can develop in frontal, ethmoid, sphenoid or maxillary sinuses. Usually preceded by severe upper respiratory tract infection or accompanied by an allergic reaction. A deflected nasal septum can lead to a "vacuum" headache originating from the frontal sinus.	Maxillary sinuses drain best when the patient lies down. The others drain best when the patient is vertical. Need ASA, acetominophen or codeine; vasoconstrictors often help. In rare cases surgery is indicated.	Nasal discharge. Low grade fever. Untreated can progress to orbital cellulitis. Pain is worse in some head positions, e.g. bending forward. Worsened by shaking the head or by percussion over the area. Photophobia, increased lacrimation, tender area at trochlea. Look for lid edema and dark circles around the eyes.
Headache with vasomotor rhinitis. (See muscle contraction headaches, sinus headaches and allergic headaches.)	Mild headache.	Frontal with nasal discomfort.	Periodic.	Hours.	Stress leading to congestion and edema of nasal mucosa without infection or allergy; has a vascular basis.	Accurate diagnosis. Symptoms can be brought on by fatigue, chilling, anxiety or anger.	May be confused with sinusitis, allergic reactions or a common cold. Lid edema is common and a few develop scotomas.

Table 22

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Pain due to middle-ear infections. (Otitis media and mastoiditis are now rare.)	Severe, stabbing, throbbing pain.	Localized, often begins in occiput then becomes frontal. A type of trigeminal neuralgia. Pain in the ophthalmic branch of CNV. Gradenigo's syndrome (the temporal syndrome) involves the petrous bone and mastoid. (See trigeminal neuralgia.)	Constant. With Gradenigo's syndrome patients have painful palsy of CNVI and ipsilateral hearing loss.	Persisting and often worse at night.	Infection, but trauma and hemorrhages are other possible causes. Gradenigo's syndrome begins as a middle ear infection but is now rarely seen. It causes palsy of CNVI or VII and sometimes CNIII in addition to a unilateral headache.	Treating the underlying cause. Need codeine every 4 hours to suppress the pain and antibiotics to control the infection. Surgery is not employed in treatment of these conditions as often as it was formerly.	Hearing loss, tinnitus, fever. Can cause facial paralysis (CNVII), often affects CNVI and rarely CNIII or IV. Can progress to meningitis. Photophobia, increased lacrimation, reduced corneal sensitivity, paralysis of lateral rectus, diplopia, esotropia.
Pain due to dental disorders, abscessed teeth, malocclusion, temperomandibular joint disorders, mandibular joint neuralgia. (See muscle tension headache, temporal arteritis and glossopharyngeal neuralgia.)	Headache is usually moderate. A referred pain. However in Costen's syndrome the pain is severe, a glossopharyngeal neuralgia. This pain seems to be supraorbital even though CNIX is involved.	Pain in ear, jaw, throat, tongue and nose. Usually unilateral, frontal or temple, less commonly in vertex or occiput and extending to the neck and shoulders. Tender area over muscles of mastication. Some have a supraorbital pain associated with branches of CNV.	Constant but worse if chewing is attempted. Limitation of jaw movement. We clench our teeth when under stress.	Often constant with some fluctuations.	Dental malocclusion, or muscle spasm associated with psychological factors. Irritation of sensory branches of CNV and later sustained muscle contraction in head and neck. (Jaw claudication often develops with temporal arteritis.)	Dental treatment. Fewer than 5% of cases are due to arthritis of these temperomandibular joints. Costen's syndrome may have a large psychological component.	Temperomandibular joint disorders are more common in older patients especially women. Some hear a clicking or grinding when the jaw is moved, many suffer impaired hearing, tinnitus, low grade fever, dizziness; rarely photophobia, excess lacrimation, conjunctival hyperemia and nystagmus.
Headaches due to combined mechanisms, mixed headaches. (These headaches blur the distinction between migraine and muscle tension headaches, however any combination of causes can occur and complicate the diagnostic problem.)	Features of two or more types. Some people appear to progress from migraine type episodes to chronic, persisting headaches.	Various. Not very informative in terms of suggesting specific causes.	Daily or irregularly or following two patterns.	Years.	About 33% of those with migraine develop a muscle tension ache too. Muscle contraction can impair blood flow and lead to a local ischemic problem or even to a rebound vascular headache.	Reducing daily use of analgesics, and trying prophylactic agents, psychotherapy and biofeedback. Avoiding situations which precipitate or aggravate the discomfort.	In about 25% of cases a headache has two or more causes, or a person has two types of headache more or less concurrently. Any type of headache can be combined with or induce depression and muscle contraction especially when the headache persists.

Table 23

Headache Class.	Pain Type	Pain Location	Pain Frequency	Pain Duration	Probable Cause	Helped by:	Comments
Headache due to refractive errors or disorders of binocularity. (Cause less than 10% of headaches.) (Possibly 1% to 3% of headaches apparently localized in the ocular region have an ocular cause.) (See Roy's ocular migraine and ophthalmoplegic migraine.)	Headache is usually dull if due to refractive problems or disorders of binocular vision.	Rarely one-sided, supraorbital, frontooccipital and temporal. Pain in the distribution of the ophthalmic branch of CNV, but may be occipital with ocular muscle imbalance.	If due to refractive problems, pain appears daily, toward end of day or after intensive near work.	Until close work is stopped or the problem is corrected.	Undercorrected hyperopia, astigmatism, or disorders of binocularity including aniseikonia. Contraction of muscles of head and neck. Squinting and frowning have been described as use of the auxiliary muscles of accommodation.	Refractive correction, visual training, absorptive lenses, prisms where needed. Be sure no organic problem exists.	Photophobia often accompanies headaches whether they are of ocular origin or not and nearly always occurs with iritis and with corneal damage.
Uveitis.	Pain is usually dull but can be marked if due to anterior uveitis (or scleritis).	Orbit, nose, eye, temples.	Worse at night except for the photophobia which is worse in bright light.	Prolonged and becoming more severe.	Primary or secondary iritis. (Scleritis or other ocular conditions can cause similar discomfort.)	Uveitis requires analgesics, cycloplegics and steroids.	Flare and cells in anterior chamber, miosis, lower IOP, photophobia. Risk of posterior synechiae and secondary glaucoma.
Glaucoma. (Older patients with chronic headache should always have their intraocular pressure measured and their visual fields assessed.)	Pain can be severe if due to angle-closure glaucoma. Referred pain.	In the area supplied by the ophthalmic branch of CNV.	Intraocular pressure tends to increase during the day so that an angle-closure attack may develop at twilight or at a movie for example, when the pupil becomes dilated.	Usually until pressure is relieved but a few have short episodes and a rare person has no pain even with very high intraocular pressure.	Glaucoma. Field loss can progress even if intraocular pressure is only slightly elevated. Field losses, fundus changes and IOP show poor correlations in some glaucoma patients.	Immediate treatment is by drugs, pilocarpine, glycerol and possibly a carbonic anhydrase inhibitor but surgery (iridectomy) is required for adequate control of angle closure type glaucoma.	With an acute attack patients have nausea, vomiting, steamy cornea, a semi-dilated pupil and reduced vision. With open-angle glaucoma symptoms are minimal but field loss develops progressively.

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NOTICE OF GENERAL BUSINESS MEETING THE CANADIAN ASSOCIATION OF OPTOMETRISTS

Friday, August 7, 1987: 9:00 am - 12:00 noon

Loyalist Room
Saint John Trade and Convention Centre

Notice is given pursuant to By-Law 34

CALL FOR RESOLUTIONS

Each resolution by an ordinary member (individual) optometrist) shall be signed by a proposer and five other ordinary members and, if by a corporate member (provincial optometric Association), shall be signed by its representative. Resolutions shall be filed with the Executive Director of CAO, Mr. Gerard Lambert, *before* the opening day of the Congress, Monday, August 3, 1987.

C.A.O. 20TH BIENNIAL CONGRESS REGISTRATION
August 4-7, 1987, Saint John, New Brunswick

Please Print or Type

OPTOMETRIST: _____
surname — first name for badge — degree — year of graduation

_____
Office address

_____
City — Prov. — Postal Code

_____
Telephone

First C.A.O. Congress? ☐ Yes ☐ No

_____
*Home address

_____
City — Prov. — Postal Code

_____
Telephone

SPOUSE/GUEST: _____
surname — first name for badge

Please check if attending:

☐ Thursday: Bus Tour to St. Andrews (Numbers will be limited)

☐ Friday: Golf

***Spouses/Guests**, please include your home address so you will be sure to receive your pre-convention information letter.

JUNIOR DELEGATES:

_____ name — age

_____ name — age

_____ name — age

_____ name — age

Babysitter required? ☐ Yes ☐ No.

Registration Fees	Postmarked by May 30	After May 30
Optometrist	$250	$300
Spouse/Guest	$225	$275
Junior	$125	$145
Preschool Daycare	$45	$45

Total Fee Enclosed: $ _____

Does anyone in your family require a special diet or have special needs?
Please specify:

Please Note: You may arrange your hotel accomodations directly with the hotel. A hotel reservation card will be mailed to you with your receipt.

Return this form with your cheque to:
C.A.O.
Suite 301, 1785 Alta Vista Dr.
Ottawa, Ontario K1G 3Y6

C.A.O. use only: Reg # _____ Fees # _____ Date _____ Cash Rec'd _____

ck rec'd _____ CN _____ Note _____

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Nova Scotia:	
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Nfld. & Quebec	1-800-565-7180
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Western Provinces	1-800-565-7166
P.E.I.:	1-902-892-2457
or write:	Visitor Services Box 940 Charlottetown, P.E.I. C1A 7M5
Car Rental:	
Budget	1-800-268-8900
Avis	1-800-268-2310
Tilden	1-800-361-5334

TRAVEL ARRANGEMENTS

Ms. Joanne Sheps, of Marlin Travel's Winnipeg office, is the official travel coordinator for this Congress. Call her — collect — at (204) 947-9870. If busy or after hours, call Air Canada direct at 1-800-361-7585, advising them that you are travelling to the CAO Congress.

Marine Atlantic Ferry to Nova Scotia
1-800-565-9470

Campground: Rockwood Park, Saint John
1-506-652-4050

Motor Home rental:
Harbour City Dodge: 1-506-634-6060
Leisure Time Sales: Derek Dobson
P.O. Box 635
Saint John, N.B. E2L 4A5
1-506-849-3363

AVIS DE L'ASSEMBLÉE GÉNÉRALE DE L'ASSOCIATION CANADIENNE DES OPTOMÉTRISTES

Le vendredi 7 août: 9h00 - 12h00

Salon Loyalist
Saint John Trade and Convention Centre

Avis donné conformément au Règlement 34

APPEL DES RÉSOLUTIONS

Chaque résolution proposée par un membre ordinaire (optométriste privé) doit être signée par celui (celle) qui la propose et par cinq autres membres ordinaires et, si elle est proposée par un membre collectif (association provinciale d'optométrie), elle doit être signée par son représentant. Les résolutions seront déposées auprès du directeur général de l'A.C.O., M. Gérard Lambert, *avant* le jour d'ouverture du congrès biennal le lundi, 3 août 1987.

20ième CONGRES BIENNAL DE L'A.C.O.
4-7 août 1987, Saint John, Nouveau-Brunswick

Prière de dactylographier ou d'écrire en lettres moulées

OPTOMÉTRISTE:

Nom — Prénom pour le porte-nom — grade — année de promotion

Adresse du bureau

Ville — Prov. — Code postal

Téléphone

*Résidence

Ville — Prov. — Code postal

Téléphone

Est-ce votre premier congrès de l'A.C.O.?
☐ Yes ☐ No

CONJOINT(E) :

Nom — Prénom

Cochez pour indiquer la participation:

☐ Jeudi: Circuit de jour en autocar à St. Andrews (participation limitée)

☐ Vendredi: Golf

***Conjoint(e):** Veuillez inscrire l'adresse de votre résidence pour recevoir l'information pré-congrès.

JEUNES:

Prénom — Âge

Prénom — Âge

Prénom — Âge

Prénom — Âge

Est-ce qu'une gardienne est nécessaire? ☐ Oui ☐ Non

Droits d'inscription:	Avant le 30 mai	Après le 30 mai
Optométriste	$250	$300
Conjoint(e)	$225	$275
Jeune	$125	$145
Garderie pré-scolaire	$45	$45

Total inclus:$ _____

Est-ce que quelqu'un dans votre famille a besoin d'une diète spéciale ou a des besoins spéciaux? Veuillez spécifier:

À noter: vous devez faire vos réservations directement avec l'hôtel. Une carte de réservation vous sera envoyée avec votre reçu.

Retournez cette formule avec votre chèque à:
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301-1785, prom. Alta Vista
Ottawa (Ontario) K1G 3Y6

Réservé à l'usage du bureau de l'A.C.O.

N° d'inscr. _____ N° des droits _____ Date _____ Argent reçu _____

Ch. reçu _____ N° de ch. _____ Remarque _____

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Ontario	1-800-565-7140
Provinces de l'Ouest	1-800-565-7166
Î.P.É:	1-902-892-2457
ou écrivez à:	Services aux visiteurs C.P. 940 Charlottetown, Î.P.É. C1A 7M5
Location de voitures::	
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Avis	1-800-268-2310
Tilden	1-800-361-5334

RÉSERVATIONS D'AVION

Mlle. Joanne Sheps, du bureau de Winnipeg de Marlin Travel, est la coordonnatrice officielle de ce congrès. Vous pouvez lui téléphoner — à frais virés — au (204) 947-9870. Si la ligne est occupée ou les bureaux sont fermés, appelez Air Canada directement au 1-800-361-7585, sans oublier de mentionner que l'objet de votre voyage est d'assister au congrès de l'ACO.

Ferry Marine Atlantic à la Nouvelle-Écosse
1-800-565-9470

Terrain de camping: Parc Rookwood
1-506-652-4050

Location de roulottes:
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Leisure Time Sales: Derek Dobson
C.P. 635 Saint John, (N.B.)
E2L 4A5 1-506-849-3363

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ANNOUNCEMENTS/NOUVELLES

COMING EVENTS

1987

May 24 - 27

Association for the Care of Children's Health (ACCH) 22nd Annual Conference
World Trade and Convention Centre
Halifax, NS

Information: ACCH
3615 Wisconsin Avenue
NW
Washington, DC
20016-3007
USA
Tel. (Canada)
1-800-565-7166
or (902)425-5781

October 22 - 25

12th Scandinavian Optometric Congress
"The Importance of Vision in Reading and Writing"
Hotel Novotel
Gothenburg, Sweden

Information: Skandinavisk Optometri Kongres
c/o Borje Zakrisson
Dragonvagen 4
S-632 33 Eskilstuna
Sweden
Tel. (016) 11 82 00

1988

April 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

April 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 25 - 28

British Contact Lens Association International Contact Lens Centenary Congress and Exhibition
London, England

Information: Conference Associates
ICLCC
27A Medway Street
London, SW1P 2BD
England
Tel. 01-222-9493

1987 OPTOMETRIST'S DESK REFERENCE — CORRECTION

Astute reviewers of the 1987 Optometrist's Desk Reference have pointed out that seventeen of Nova Scotia's optometrists have vanished at the end of p.117. A typesetting computer's glitch inadvertently located them on p.92, directly after the Winnipeg, Manitoba listings.

CAO apologizes for any inconvenience caused to the practitioners of either province.

VISION CARE NEWS/ACTUALITÉ OCULO-VISUELLE

CAO President Dr. Scott Brisbin fields one of many calls received during the Sunday, March 1 "Dial-the-President" Day at the national office.

Dial-the-President a Ringing Success

Successful enough "to encourage a repeat next year" is CAO President Dr. Scott Brisbin's assessment of the Association's first ever "Dial-the-President" Day. Dr. Brisbin spent virtually the entire day, Sunday, March 1, in the Conference Room at the new CAO offices fielding questions and comments from practitioners across Canada.

In a post-event analysis, Dr. Brisbin said that he was very pleased with the response and the range of topics discussed by the callers which, he said, indicated a widespread interest in the program carried out by the national Association.

In fact, he reported that he was still receiving calls — at his Edmonton office (!) — up to a week after the event from members who had either forgotten to call or had been unable to get through during the scheduled time.

CooperVision Distributes Hefty UV Information Kit to Media

Canadians Unaware of Ultraviolet Radiation Dangers is the headline on the lead-off news release in a package of information developed by a Toronto communications company for CooperVision in support of its Permaflex UV Lens.

Also included in the media kit are "backgrounders" on UV Radiation (which begins, "Harmful ultraviolet radiation known to cause sunburns, induce skin cancer and age skin, is strongly suspected of damaging other body parts as well."); How the Eye Works; a Corporate Backgrounder on CooperVision; its Permaflex UV Lens and a supplemental "Product Fact Sheet" also on the Permaflex UV which advises us that the lens absorbs UV radiation, converts it to heat and transmits the heat back into the air".

Further information is available from your local CooperVision contact or directly from CooperVision Inc., 100 McPherson Street, Markham, ON, L3R 3V6. Tel.(416)475-8555.

Dr. K.S. Chhatwall (R) accepts one of four service awards presented by Dr. Jacob Sivak, Director, Associate Dean of Science for Optometry, University of Waterloo.

Waterloo School Awards Presented

Four long-time teachers in the School of Optometry, University of Waterloo's clinic were honoured recently at a special Awards Assembly. Recognized for "Dedicated Teaching" were Drs. R.R. Chen, K.S. Chhatwall, G.A. Grant and R. Saari, each of whom received a special commemorative certificate in appreciation of his services to the School.

SOLA Technical Assistance Hotline

SOLA Optical USA Inc. has announced the inauguration of a new Technical Assistance Hotline to answer questions regarding the dispensing of any of its products, including the VIP progressive addition lens.

Unfortunately, in spite of the fact that the *CJO • RCO* was provided a news release announcing the service, it is accessible only through an "800" number that appears to cut off at the Canada/US border (or at least did when access was attempted from our Ottawa offices).

In the meantime, further information about SOLA products can be requested through regular postal and telephone routes at the following address:

SOLA Optical USA, Inc.
3600 Lakeville Highway
Petaluma, CA
94952
USA
Tel.(707)763-9911

N and N / Wetzel Merger Creates New CL Company

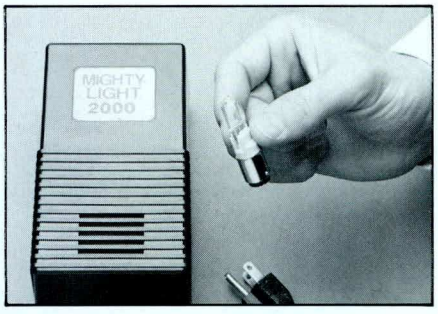
NAVCO (the North American Vision Company) is the name of a new firm formed by the merger of N and N Optical of Canada and Wetzel Contact Lens.

VISION CARE NEWS/ACTUALITÉ OCULO-VISUELLE

In announcing the merger recently, NAVCO's VP-Finance, Peter Snook advised that its intent is to provide even greater access to Canadian markets.

Further information is available from NAVCO's public relations agency:

Matthews Associates
Suite 1018, 603 Stewart Street
Seattle, WA
98101
USA
Tel. Carole Holm (206)340-0680

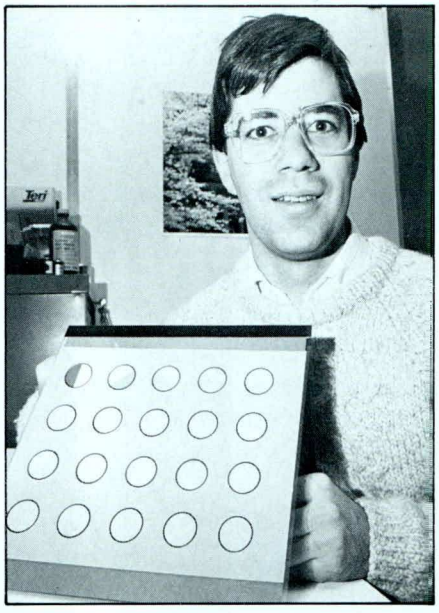


Mighty Light 2000 Illumination System

Medical Light Source, Inc. has announced a new product designed to improve illumination for standard chart projectors. According to a news release accompanying the announcement, the Mighty Light 2000 will "prevent 40 future exam interruptions and reduce bulb costs by 60%". Its quartz halogen bulb is rated for 2000 hours, 40 times longer than the older style of incandescent projector bulbs.

The complete system, which includes a low voltage power supply and a "uniquely engineered" quartz halogen bulb with a "Twist Assist" ceramic collar to save seared fingers, carries a suggested retail price of $129.95 (US$), although the company also promotes a trade-in program which could drop the price to as low as $89.95 (US$). According to the material received by the *CJO •RCO*, the system will be available for a limited time for a free trial period from:

Medical Light Source, Inc.
3133 North El Paso Street
Colorado Springs, CO
80907-5414
USA
Tel.(303)577-4931



Australian Eye Chart Measures Contrast Detection

An Australian optometrist, Dr. Jos Verbaken of Melbourne, has developed a low cost eye chart which measures a patient's ability to detect contrast.

Introduced at the October, 1986 Australian Optometrical Association Congress, Dr. Verbaken's **Melbourne Edge Test** (MET) consists of a card with 20 circles, one inch in diameter, each with a different semi-circular contrast ranging from 80 to 0.1 percent.

Says Dr. Verbaken, the MET chart "will be useful for screening vision abnormalities" like the glaucoma/diabetes link and will assist in early screening for particular diseases such as multiple sclerosis.

Dr. Verbaken holds the copyright on the chart, which he expects to market for "between $300 to $500" (because of the expensive photo-specific reproduction process required to produce the card).

Further information is available from: Dr. Jos Verbaken, 21 Waterloo Street, Carlton, Victoria 3053, Australia. Tel.(03) 347-0075.

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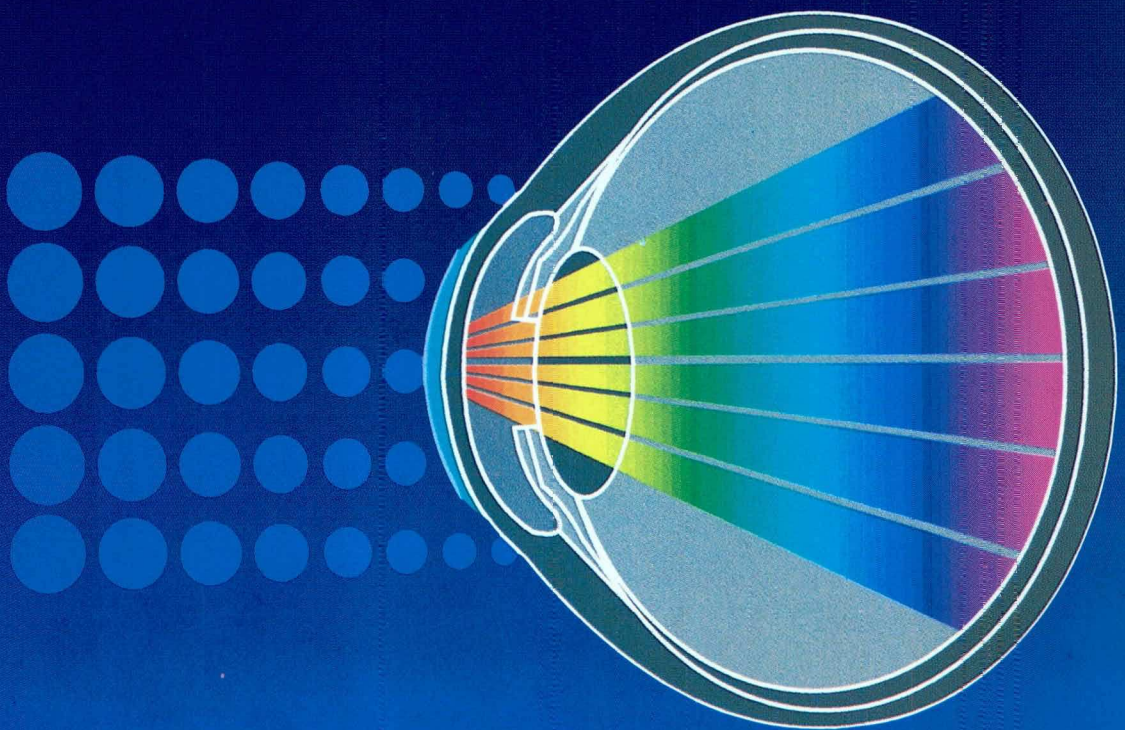

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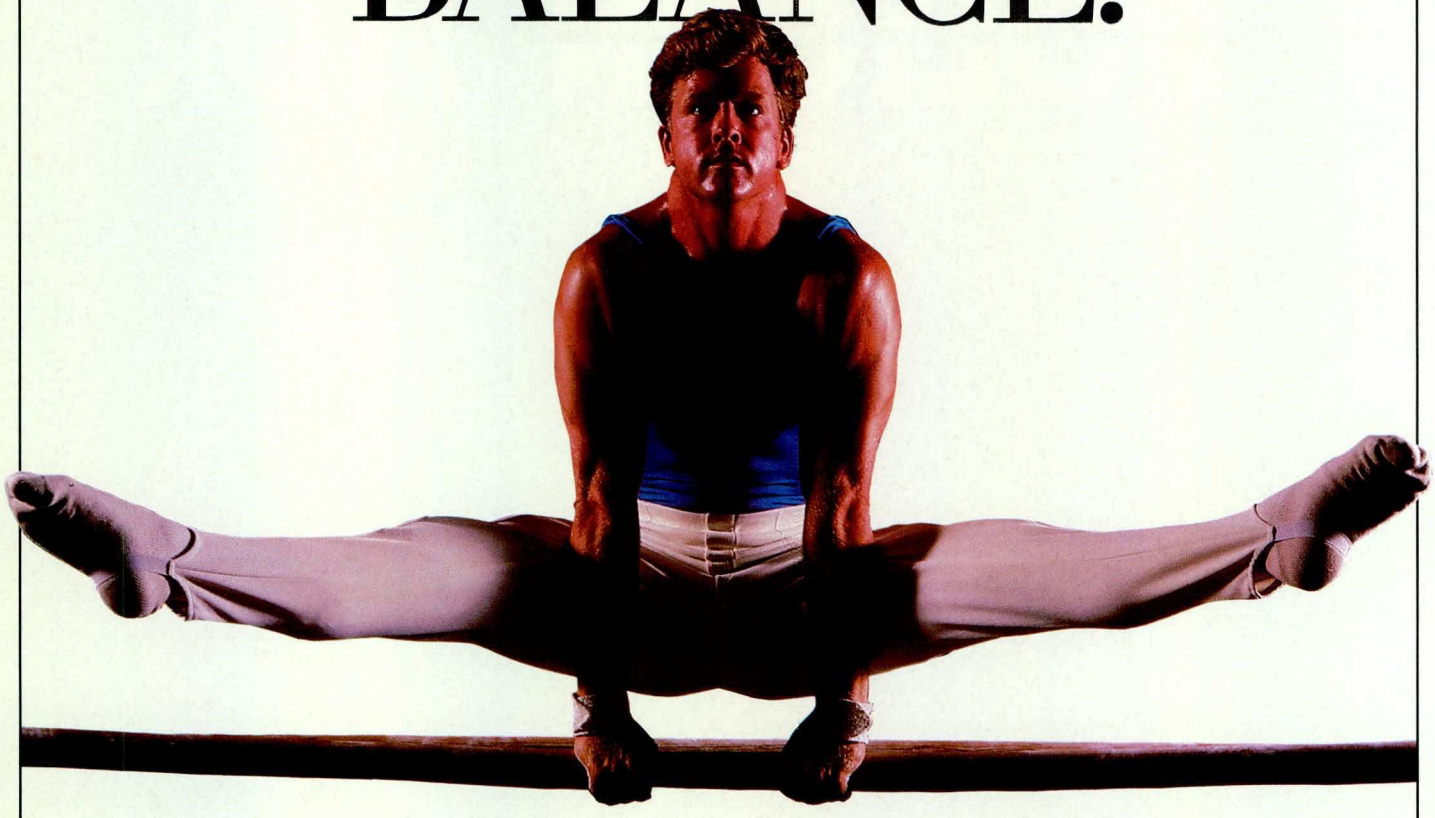
BALANCE.



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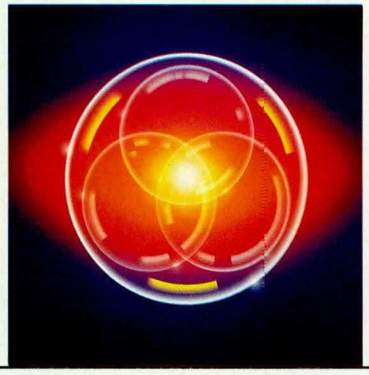

In addition, Equalens offers greater oxygen permeability and comfort than extended-wear soft lenses... with no compromise in visual acuity. And Equalens is the first RGP to feature an ultraviolet light absorber to protect the delicate structure of the eye.

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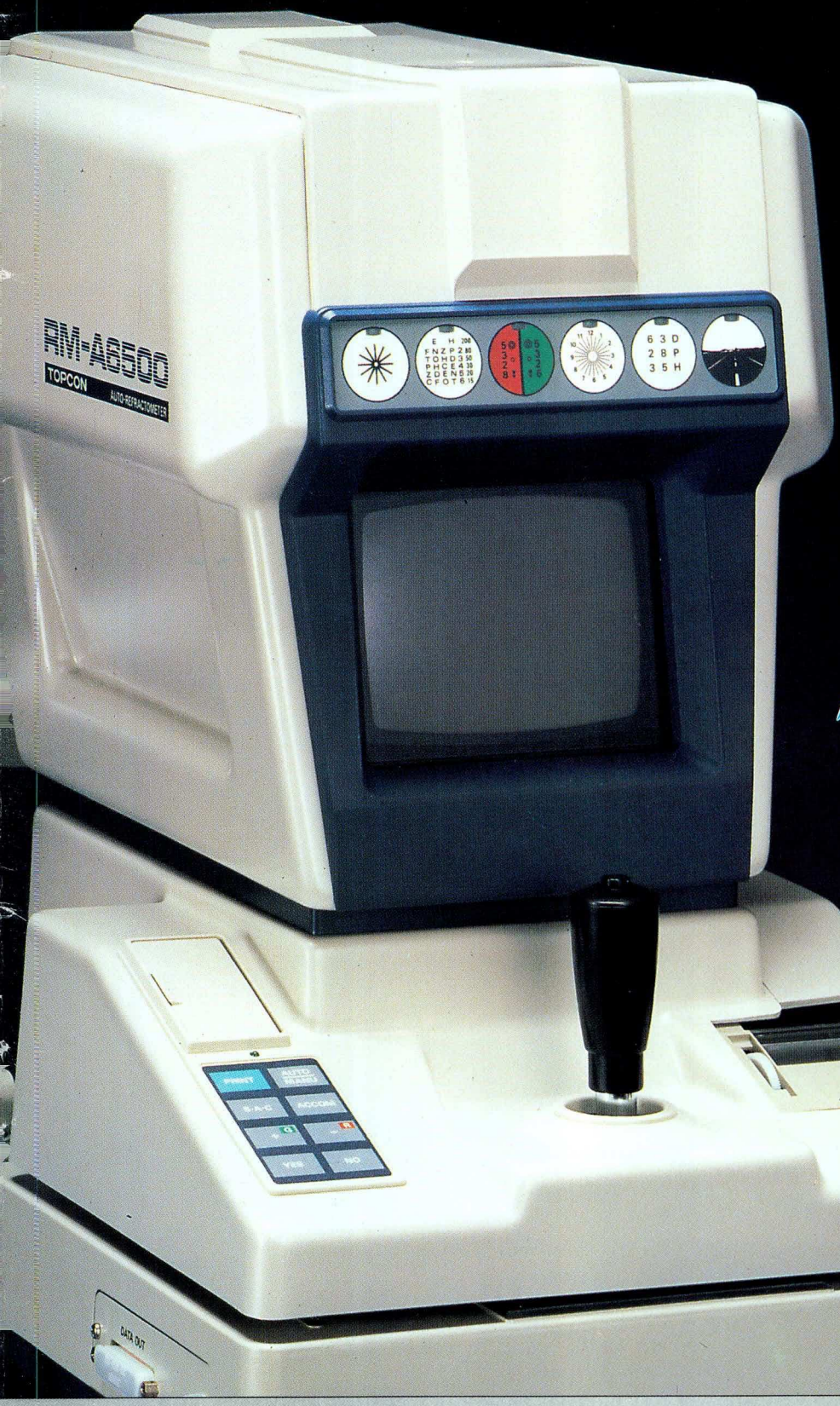

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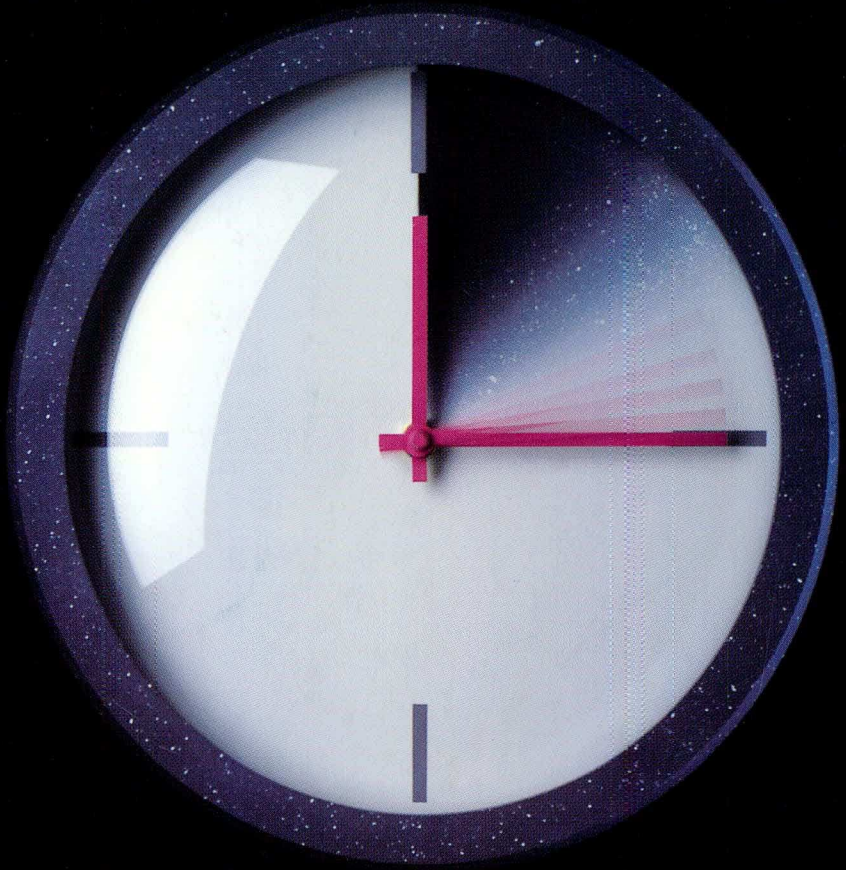

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