AN INTERNATIONAL SYMPOSIUM

Crowne Plaza Hotel–Brugge, Belgium

Honoring Dr. Charles L. Schepens

July 5-8, 2003

Presented by the Schepens International Society

Sponsored by the Truman Medical Centers, Kansas City, Missouri

RETINA 2003: Back to Belgium
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MESSAGE FROM THE PRESIDENT

Binevenue! Welcome! Welkom! Willkommen!

On behalf of the Schepens International Society, welcome to our ninth biennial symposium. Our theme “Back to Brugge” honors our founding president, Dr. Charles L. Schepens. Dr. Schepens is a teacher, mentor and friend to many of us. His teachings have touched countless lives throughout the world. It is because of his commitment to education that our Society was formed. It is through the desire that he instilled in all of us that we continue to share our knowledge around the globe.

I want to acknowledge and thank Dr. J. Sebag, our program chair, and his committee for organizing an excellent curriculum and outstanding faculty of speakers. Our mission is to share knowledge with young ophthalmologists who are in training. It is through the dedication of members like Dr. Sebag that we fulfill that mission.

But sharing our clinical and scientific knowledge is only part of what our biennial symposia offer. They provide us an occasion to renew old friendships and make new ones. They are a way to develop connections with colleagues so that we can communicate and share our knowledge on a more frequent basis. They also offer us opportunities to view cultures and visit cities all around this amazing world in which we live.

Thank you for sharing your time with us, and welcome “Back to Brugge”.

The Belgian motto is “Union makes strength.” Let us all participate in the meeting of the Schepens International Society in Brugge, Belgium. Let us learn of the new experiences and the recent knowledge acquired by our European colleagues, and share with them much of what we have learned recently in America. This will allow all of us to move into the 21st century with increased strength and confidence.

Dr. Glenn L. Wing
Since its inception, the Schepens International Society has met in several world capitals and has most recently journeyed from the Grand Teton mountains to the Nevada desert. We now find ourselves going back to Belgium. This is not only to honor the namesake of our organization by visiting the country of his birth, but also to convene in the splendor of a very special part of the world.

The city of Brugge offers the Schepens International Society and its guests a unique venue for our ninth meeting. The ambiance of this ancient cultural center will be conducive to both academic objectives and social aspirations for this meeting.

I am greatly indebted to the members of the program committee and the outstanding speakers who have agreed to form the faculty of this meeting. This group represents a marvelous blend of European, Japanese, North and South American experts, all leaders in the field. Their participation in this meeting not only creates an agenda of superb quality and relevance to advancing vitreo-retinal eye care, but also embodies the spirit of what our civilization strives to achieve - international cooperation and collegiality orchestrated for the purpose of education and progress to benefit people around the world. Thank you for being part of this international venture, as we go

"Back to Belgium".

BRUGGE PROGRAM COMMITTEE:

Jerry Sebag, MD, Chair - California, USA
Thierry Verstraeten, MD - Pennsylvania, USA
Jean Jacques DeLaey, MD - Ghent, Belgium
Prof. Peter Kroll - Marburg, Germany
Carl Claes, MD - Antwerp, Belgium
Prof. Akitoshi Yoshida - Asahikawa, Japan
Prof. Gabriel Coscas - Paris, France
Bart LaFaut, MD - Brugge, Belgium
Prof. Gisele Soubrane - Paris, France
Glenn Wing, MD – Florida, USA
Nelson Sabates, MD – Missouri, USA
SATURDAY, JULY 5, 2003

Pre-Registration ......................................................... 15:00
Welcome Cocktail Reception-Sint Donaas Room ............ 17:00-19:00

SUNDAY, JULY 6, 2003

Registration ............................................................... 6:45
Continental Breakfast-Restaurant De Linde ............... 6:45-7:45
Exhibits Burgh IV, V ..................................................... 7:00-17:15
Welcome—Burgh I, II, III ............................................... 7:45
Leslie Nesmith Lecture (Dr. Paul Bishop) ..................... 8:00
Vitreous ................................................................. 9:00
Vitreo-Retinal Interface ............................................. 10:30
Business Meeting ...................................................... 11:30
Lunch ................................................................. 12:00
Vitreo-Retinal Surgery I ............................................ 13:30
Vitreo-Retinal Surgery II ........................................... 15:15
Gala Dinner—Belfry Tower ........................................... 19:00

MONDAY, JULY 7, 2003

Registration ............................................................... 7:00
Continental Breakfast-Restaurant De Linde ............... 7:00-8:00
Exhibits Burgh IV, V ..................................................... 7:00-16:45
ARMD Pathogenesis & Clinical Diagnostics ................. 8:00
Paul Kayser Award I (Dr. Eugene de Juan) ................. 10:10
ARMD Therapeutics ................................................... 11:10
Lunch ................................................................. 12:00
Medical Retina/Uveitis ............................................. 14:15
Special Presentation to Dr. Schepens-Town Hall .......... 17:30
Dinner—Old St. John Hospital .................................... 19:00

TUESDAY, JULY 8, 2003

Registration ............................................................... 7:00
Continental Breakfast-Restaurant De Linde ............... 7:00-8:00
Exhibits Burgh IV, V ..................................................... 7:00-13:00
Diabetes ................................................................. 8:00
Paul Kaiser Award II (Prof. Wallace Foulds) ............... 10:30
Vitreo-Retinal Surgery III ........................................... 11:30
Adjournment ........................................................... 13:00
**WELCOME RECEPTION**

*Saturday, July 5, 2003*
Saint Donaas Room, Crowne Plaza Hotel . . . . . . . . . . . . . . . . . . . . 17:00-19:00

**GALA DINNER**

*Sunday, July 6, 2003*
The Belfry Tower–13th Century Rodenbach Room . . . . . . . . . . . . 19:00-22:00

**RECEPTION HONORING DR. CHARLES L. SCHEPENS**

*Monday, July 7, 2003*
Town Hall . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 17:00-17:45

**DINNER**

*Monday, July 7, 2003*
Old St. John Hospital including a tour of the Museum . . . . . . . . . . . . 19:00-22:00

**TOURS**

**TOUR I—INCLUDING LUNCH**

*Sunday, July 6, 2003*
Basilica of the Holy Blood
Medieval Stock Exchange
St. Jacques Church and Groeninge Museum . . . . . . . . . . . . . . . . . . . . 10:00-15:30
(Flemish Paintings)

**TOUR I—INCLUDING LUNCH**

*Monday, July 7, 2003*
Lake of Love tour through old Brugge canals.
Gruuthus Museum
Princely Beguinage of the Vineyard
Michelangelo’s Virgin and Child . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 10:00-15:30
SCHEPENS INTERNATIONAL SOCIETY PERMANENT ENDOWMENT FUND

The SIS Permanent Endowment Fund originated in 1992 to honor Dr. Charles Schepens. The goal is to raise $1 million in order to guarantee the perpetuity of the Society and its educational activities. As of December 31, 2002, the principal investment account was valued at $607,671. The corpus will always remain intact; only the interest earned will be used to help fulfill the Society motto:

“A WAY OF SHARING KNOWLEDGE”

This is being accomplished by providing travel scholarship so that young ophthalmologists in training can attend the biennial meetings. So far the Permanent Endowment Fund has generated income of $135,209, which provided 110 full meeting scholarships from 1995 through 2001.

The membership is to be complimented for supporting this worthwhile project with pledges and donations. However, we cannot rest on our laurels if we are to reach our financial goal. Each member is challenged to continue supporting the Permanent Endowment Fund.

NO GIFT IS TOO SMALL – OR TOO LARGE
The LESLIE NESMITH LECTURESHIP WAS ESTABLISHED IN 1998 IN MEMORY OF DR. LESLIE NESMITH and in appreciation for his generous contribution to the Permanent Endowment Fund. This lectureship features some of the most outstanding research efforts world-wide. The Leslie Nesmith, MD Lectureship is presented biennially at SIS meetings. For 2003, the featured presenter is Paul Bishop, PhD.

Dr. Paul Bishop qualified from Nottingham University Medical School, England in 1983. He subsequently specialized in ophthalmology and undertook most of his training in Manchester. In 1998, he was appointed as a Consultant Ophthalmologist at Manchester Royal Eye Hospital, specializing in medical retina. While undertaking clinical training he became increasingly interested in basic science and completed a PhD in Biochemistry in 1993. Since 1991, he has held consecutive Wellcome Trust funded research fellowships, culminating in 1999 when he became the first ophthalmologist to be awarded a Wellcome Trust Senior Research Fellowship in Clinical Science. Dr. Bishop's main research interest is the molecular basis of vitreous structure and vitreoretinal disease. Currently he is a Principal Investigator at the internationally renowned Wellcome Trust Centre for Cell-Matrix Research, University of Manchester.
Awards and Lectureships

THE PAUL KAYSER INTERNATIONAL AWARD OF MERIT IN RETINAL RESEARCH

Awarded his MB ChB degree in 1946, Wallace Foulds trained in Moorfields Eye Hospital in London and held research appointments in the Institute of Ophthalmology of London University and a clinical appointment as Consultant Ophthalmologist at Addenbrooke’s Hospital, Cambridge and Honorary Lecturer in the University of Cambridge. In 1964, he was appointed to the Tennent Chair of Ophthalmology in the University of Glasgow. As Tennent Professor, Wallace Foulds held a busy clinical and administrative position but nevertheless continued with his personal research in the laboratories and research clinics of the Tennent Institute.

During his tenure as the Tennent Chair, he fostered interdisciplinary research in many basic science and clinical departments in the University of Glasgow and other Institutions. Throughout his career he has always recognized the importance of developing the interface between basic science and clinical medicine and much of his research work has been at this interface. Largely because of a long and productive research co-operation with a number of Departments in Strathclyde University, Wallace Foulds was awarded an Honorary DSc in that University in 1991. During his career he has published close to 200 scientific papers and 22 chapters in books. He has given a large number of invited and named lectures.

In addition to his headship of an active University Department, and the clinical care of patients, Wallace Foulds has been active in national and international professional bodies. He held office as the first and Founding President of the Royal College of Ophthalmologists. Wallace Foulds has since been largely responsible for the planning and establishment of the Singapore Eye Research Institute (SERI) and after a period as Co-Director is now Senior Consultant in the Institute where he is responsible for Retina research. He is also currently setting up a collaborative study between SERI and the University of Glasgow to develop the use of computer-aided artificial intelligence in the identification of sight-threatening diabetic retinopathy. Wallace Foulds is also involved in an experimental study in Singapore to develop a pig model of myopia to allow further basic research into the etiology of this condition, particularly to investigate the role of retinal cell signaling in the genesis of myopia and develop therapy based upon image modification. A further study aims to develop an improved model of subretinal neovascularization to allow the testing of new treatment modalities being investigated. Lastly, recent work has shown the practicability of the supercheroidal route for such drug delivery.
His understanding of retinal diseases, his ability to discern the benefits and feasibility of potential treatments, and his ability to forge the necessary partnerships to fund and accomplish complex research projects are key elements contributing to his successful research efforts.

In particular, Dr. de Juan is known for pioneering the macular translocation surgery, for creating the Microsurgery Advanced Design Laboratory (MADLAB), and for his significant role in the ongoing efforts to develop a retinal implant. The benefit of retinal translocation, for patients meeting the appropriate criteria, is an opportunity to restore visual acuity to the level required for reading and driving. Improvement in vision is not achievable with any other current treatment for CNV. The MADLAB, currently located at Doheny Retina Institute, is dedicated to improved patient care through the design and development of innovative microsurgical devices and techniques. Created more than a decade ago and implemented with great effectiveness at other institutions, the MADLAB has produced 24 patents and more than 65 inventions licensed by medical device companies. Dr. de Juan and the MADLAB have recently developed a new standard in vitreous surgery, “25 gauge transconjunctival sutureless vitrectomy.” Dr. de Juan is also co-inventor of the intraocular retinal prosthesis and performed seminal experiments demonstrating that the sensation of vision can be produced by focal electrical stimulation of the retina of a totally blind RP patient. These experiments laid the foundation for the burgeoning field of retinal prosthesis that in ten short years has gone from a far-fetched concept to human clinical trials evaluating a prototype device. Dr. de Juan continues to shepherd this project in a pivotal advisory role.
MISSION STATEMENT OF THE SCHEPENS INTERNATIONAL SOCIETY

Schepens International Society is comprised of ophthalmologists who are fully trained in the field of vitreo-retinal diseases. Members recognize that they are extremely fortunate to have received such training when many young ophthalmologists are lacking the means or have no access to adequate training.

Therefore, members of SIS want to share their knowledge with young ophthalmologists who have no access to modern training. The Society will carry out its mission by holding periodic meetings in various locations of the world. Knowledge will be disseminated by paper presentations, scientific exhibits, posters, and courses. SIS will make every effort to provide fellowships to meritorious candidates for future training.

The Society will foster the development of friendly relationships with ophthalmologists in all parts of the world. It will also develop a spirit of cooperation and mutual support between the society’s members.

INFORMATION ABOUT MEMBERSHIP IN THE SCHEPENS INTERNATIONAL SOCIETY

Fellows of the Schepens Eye Research Institute (SERI) are automatically invited upon completion of their fellowship training.

SIS Executive Committee members may extend an invitation(s) for membership. Executive Committee approval must be unanimous. This includes regular active members and honorary members.

An applicant must have completed a vitreo-retinal fellowship, submit a current curriculum vitae and one letter of recommendation from the chairman of their fellowship, a member of Schepens International Society or a colleague.

If a member has not paid dues for three years, the treasurer will send a letter and notice of delinquency. If there is no response, the member will be automatically dropped from the membership.

To receive a membership application or for further SIS information, contact:

Patricia Wilson
Executive Secretary, SIS Administrative Office
10611 Piping Rock Lane,
Houston, TX 77042
PHONE: (713) 798-3276
FAX: (713) 798-7848
EMAIL: psw@bcm.tmc.edu
EXECUTIVE COMMITTEE

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Glenn Wing, MD .......... President
Fort Myers, FL
Rand Spencer, MD .......... Past President
Dallas, TX
Nelson Sabates, MD .......... Vice President
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William Tasman, MD
Philadelphia, Pennsylvania

Prof. Marie-Jose Tassignon
Edegem, Belgium

Hiroko Terasaki, MD
Nagoya, Japan

Prof. Akitoshi Yoshida
Asahikawa, Japan
CME CREDITS
This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of Truman Medical Centers and the Schepens International Society. Truman Medical Centers is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Truman Medical Centers designates this educational activity for a maximum of 17 category 1 credits toward the AMA Physician’s Recognition Award. Each physician should claim only those credits that he/she actually spent in activity.

The American Medical Association has determined that non-US licensed physicians who participate in this CME activity are eligible for AMA PRA category 1 credit.

PROGRAM OBJECTIVES
1. Recognize various diseases and apply new advances in the treatment of vitreo-retinal diseases
2. Know the latest approaches for vitreo-retinal diseases
3. Acquire skills to improve the diagnosis and management of vitreo-retinal diseases
4. Identify diseases commonly encountered in a clinical retina practice

VERIFICATION OF ATTENDANCE
As the accredited sponsor of this CME Symposium, The Truman Medical Centers is required to verify physicians’ attendance in order to award CME credits.

Meeting participants desiring CME Credits for this symposium must sign in at the registration desk on Sunday, July 6, Monday, July 7 and Tuesday, July 8, 2003. This documentation will verify attendance and determine the number of credit hours to be awarded to an individual.
CME CERTIFICATES
Information for the CME certificates will be gathered at the meetings and mailed to each participant after the meeting is over.

COURSE EVALUATION
A course evaluation will be available at the registration desk on each day of the meeting.

To assess the effectiveness of this Symposium in meeting the course objectives and needs of meeting participants, please fill out the evaluation form and return it to a staff member.

This information will be used in planning future CME activities.

DISCLOSURE OF FINANCIAL RELATIONSHIPS
The ACCME requires disclosure of all significant financial or other relationships that may exist between the manufacturers/suppliers of commercial products/services and the sponsors/faculty of this course. At the time of the activity and/or over the past twelve (12) months, the following relationships are/were in existence:

As a sponsor accredited by the Accreditation Council on Continuing Medical Education (ACCME), Truman Medical Center’s Department of Continuing Medical Education is charged with ensuring balance, independence, objectivity, and scientific rigor in all its continuing medical education programs. All faculties participating in a sponsored activity are expected to disclose to the audience any significant financial interest or other relationships with the manufacturer(s) of any commercial product(s) and/or provider(s) of commercial services discussed in an educational presentation. The disclosure is a way of providing the audience with information regarding any relationship that might influence the presentation.

These disclosures are a way to provide attendees with information on which they can make their own judgments. It remains for the audience to determine whether the speaker’s interest or relationship may influence the presentation with regard to exposition or conclusion. The presence of such relationships does not, in and of itself, necessarily imply bias or decrease the value of the presentation.
As a sponsor accredited by the Accreditation Council on Continuing Medical Education (ACCME), Truman Medical Center’s Department of Continuing Medical Education is charged with ensuring balance, independence, objectivity, and scientific rigor in all its continuing medical education programs. All faculties participating in a sponsored activity are expected to disclose to the audience any significant financial interest or other relationships with the manufacturer(s) of any commercial product(s) and/or provider(s) of commercial services discussed in an educational presentation. The disclosure is a way of providing the audience with information regarding any relationship that might influence the presentation.

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The Commercial Support Standards of the ACCME require that presentations be free of commercial bias and that any information regarding commercial products/services be based on scientific methods generally accepted by the medical community. If a presentation has discussion of unlabeled/investigational use of a commercial product, that information must be disclosed to the activity participants.

Each faculty member has been asked to complete and return a form disclosing commercial interests they have with the manufacturer(s) of any commercial product(s) or provider(s) of commercial services. The information contained below has been compiled from the returned responses.

<table>
<thead>
<tr>
<th>Speaker</th>
<th>Name of Commercial Entity/Type of Relationship</th>
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<tbody>
<tr>
<td>Mikki Arai, M.D.</td>
<td>Disclosure information will be provided prior to this presentation.</td>
</tr>
<tr>
<td>Adiel Barak, M.D.</td>
<td>Dr. Barak has nothing to disclose.</td>
</tr>
<tr>
<td>Prof. Udo Bartsh</td>
<td>Professor Barytsh has nothing to disclose.</td>
</tr>
<tr>
<td>Prof. Susanne Binder</td>
<td>Professor Binder has nothing to disclose.</td>
</tr>
<tr>
<td>Prof. Alan Bird</td>
<td>Professor Bird has nothing to disclose.</td>
</tr>
<tr>
<td>Paul Bishop, PhD</td>
<td>Dr. Bishop has nothing to disclose.</td>
</tr>
<tr>
<td>David Charteris, M.D.</td>
<td>Dr. Charteris has nothing to disclose.</td>
</tr>
<tr>
<td>Carl Claes, M.D.</td>
<td>Disclosure information will be provided prior to this presentation.</td>
</tr>
<tr>
<td>Prof. Gabriel Coscas</td>
<td>Disclosure information will be provided prior to this presentation.</td>
</tr>
<tr>
<td>Eugene de Juan, Jr., M.D.</td>
<td>Dr. de Juan’s presentation(s) will include discussion of commercial products. Dr. de Juan will also be discussing the use of a product that has not been approved by the United States Food and Drug Administration for the purpose to be discussed. Dr. de Juan receives grant/research support from Bausch and Lomb. He is also a consultant for Bausch and Lomb. He is a stockholder of Innorx and receives other financial or material support from Bausch and Lomb and Innorx.</td>
</tr>
<tr>
<td>Prof. Marc de Smet</td>
<td>Disclosure information will be provided prior to this presentation.</td>
</tr>
<tr>
<td>Stephen Dunker, M.D.</td>
<td>Dr. Dunker has nothing to disclose.</td>
</tr>
<tr>
<td>Andrew W. Eller, M.D.</td>
<td>Dr. Eller has nothing to disclose.</td>
</tr>
<tr>
<td>Prof. Wallace Foulds</td>
<td>Professor Foulds has nothing to disclose.</td>
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</table>
**Symposium Evaluation & Speaker Disclosure**

**Professor Soubrane**

Professor Soubrane’s presentation(s) will include discussion of commercial products. In addition, Professor Soubrane will be discussing a product that has not yet been approved by the United States Food and Drug Administration for the purpose to be discussed. Professor Soubrane receives grant/research support from Novartis and Alcon. He is also on the speakers’ bureau of Novartis.

**Rand Spencer, M.D.** Disclosure information will be provided prior to this presentation.

**William Tasman, M.D.** Dr. Tasman has nothing to disclose.

**Prof. Marie-Jose Tassignon** Dr. Tassignon has nothing to disclose.

**Hiroko Terasaki, M.D.** Dr. Terasaki has nothing to disclose.

**Prof. Akitoshi Yoshida** Professor Yoshida has nothing to disclose.

**Disclosure information will be provided prior to this presentation.**

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**Lamminier Francois, M.D.** Disclosure information will be provided prior to this presentation.

**John Gonder, M.D.** Disclosure information will be provided prior to this presentation.

**Anne C. Gribomont, M.D.** Dr. Gribomont has nothing to disclose.

**Gregory Hageman, PhD** Dr. Hageman’s presentation will not include discussion of a commercial product. Dr. Hageman receives grant/research support from Alcon, one of the commercial supporters for this conference.

**Sachiko Hamada, M.D.** Disclosure information will be provided prior to this presentation.

**Carlos H. Garcia, M.D.** Disclosure information will be provided prior to this presentation.

**Tatsuo Hirose, M.D.** Dr. Hirose has nothing to disclose.

**Prof. Eva Kohner** Professor Kohner’s presentation(s) will include discussion of commercial products. Professor Kohner will also be discussing the use of a product that has not been approved by the United States Food and Drug Administration for the purpose to be discussed. Professor Kohner receives grant/research support from Eli Lilly and CO. She is also a consultant for their European and British Advisory Board Members.

**Prof. Ingrid Kreissig** Professor Kreissig has nothing to disclose.

**Prof. Peter Kroll** Disclosure information will be provided prior to this presentation.

**Dr. Janet Liversidge** Disclosure information will be provided prior to this presentation.

**Anad Loewenstein, M.D.** Dr. Loewenstein has nothing to disclose.

**J. Wallace McMeel, M.D.** Dr. McMeel’s presentation(s) will include discussion of commercial products. Dr. McMeel receives grant/research support from Zeneca.

**Prof. Yozo Miyake** Professor Miyake has nothing to disclose.

**P. Andrew Pearson, M.D.** Dr. Pearson’s presentation(s) will include discussion of commercial products. Dr. Pearson will also be discussing the use of a product that has not been approved by the United States Food and Drug Administration for the purpose to be discussed. Dr. Pearson receives grant/support funds from Control Delivery Systems. He is a consultant for Bausch and Lomb and Control Delivery Systems. He is a stockholder of Control Delivery Systems and receives other financial or material support from patents.

**Prof. Gisbert Richard** Professor Richard has nothing to disclose.

**Alvaro Rodriguez, M.D.** Dr. Rodriguez has nothing to disclose.

**Maria B. Rougier, M.D.** Dr. Rougier has nothing to disclose.

**John Sarks, M.D.** Dr. Sarks has nothing to disclose.

**Shirley Sarks, M.D.** Ms. Sarks has nothing to disclose.

**Mateusz Scibor, M.D.** Disclosure information will be provided prior to this presentation.

**Jerry Sebag, M.D.** Dr. Sebag has nothing to disclose.

**Prof. Jean Louis Selam** Dr. Selam has nothing to disclose.

**Prof. Gisele Soubrane** Professor Soubrane’s presentation(s) will include discussion of commercial products. In addition, Professor Soubrane will be discussing a product that has not yet been approved by the United States Food and Drug Administration for the purpose to be discussed. Professor Soubrane receives grant/research support from Novartis and Alcon. He is also on the speakers’ bureau of Novartis.

**Rand Spencer, M.D.** Disclosure information will be provided prior to this presentation.

**William Tasman, M.D.** Dr. Tasman has nothing to disclose.

**Prof. Marie-Jose Tassignon** Dr. Tassignon has nothing to disclose.

**Hiroko Terasaki, M.D.** Dr. Terasaki has nothing to disclose.

**Prof. Akitoshi Yoshida** Professor Yoshida has nothing to disclose.
SYMPOSIUM UNDERWRITERS

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Pre-registration .......................... 15:00 - 17:00
Welcome Cocktail Reception .................. 17:00 - 19:00

CROWNE PLAZA HOTEL—Ground Floor
SUNDAY, 7:45-12:00
Registration & Continental Breakfast .......................... 6:45-7:45
View Exhibits ................................................................ 6:45-7:45
Presidential Welcome .................................................. 7:45
  Glenn Wing, MD, President
Program Chairman Welcome
  Jerry Sebag, MD, FACS, FRCOphth, Program Chairman

LESLEY NESMITH AWARD LECTURE
The Gel State of Vitreous from a Molecular Perspective .......... 8:00-9:00
  Paul Bishop, PhD, FRCS, FRCOphth

VITREOUS
Moderators: Prof. A. Yoshida and J. Sebag, MD
What is the Vitreous For? .............................................. 9:00-9:20
  Prof. Wallace Foulds
Pharmacologic Vitreolysis ........................................... 9:20-9:40
  Jerry Sebag, MD
Pharmacologic Vitreolysis Using Microplasmin ................. 9:40-10:00
  Prof. Marc De Smet

BREAK/EXHIBITS OPEN .................................................. 10:00-10:30

VITREO-RETINAL INTERFACE
Moderators: J Sebag, MD and Prof. A. Yoshida
Peripheral Vitreo-Retinal Interface .............................. 10:30-10:50
  Stephan Dunker, MD
Aging of the Vitreous Base .................................... 10:50-11:10
  Paul Bishop, PhD
Characterization and Histogenesis .............................. 11:10-11:30
  Andrew Eller, MD
of the Internal Limiting Lamina

BUSINESS MEETING .................................................. 11:30-12:00

LUNCH/EXHIBITS OPEN .................................................. 12:00-13:30
The gel state of the vitreous is maintained by a diluted network of thin collagen fibrils. With aging, there is progressive aggregation of these fibrils into thick fibres leading to a redistribution of collagen within the vitreous cavity and consequent liquefaction. The individual collagen fibrils contain a core of fibrillar collagen (mainly type II collagen) that is coated with covalently bound type IX collagen and other non-covalently bound molecules. Early work showed that type IX collagen possesses a chondroitin sulphate side-chain that could contribute towards spacing apart the collagen fibrils. Now we have shown that there is a dramatic loss of type IX collagen from the fibril surfaces with aging and a corresponding increase in the surface exposure of “sticky” type II collagen: thus predisposing to fibrillar aggregation and hence vitreous liquefaction. Analyses of non-covalently bound molecules on the fibril surface revealed that the predominant component is a novel glycoprotein that we have called opticin. Opticin is likely to contribute towards the shielding of type II collagen from the fibril surfaces. Additionally, we have recently shown that opticin interacts with cells through integrins and suggest that these interactions are important in vitreoretinal disease.
WHAT IS THE VITREOUS FOR?

Prof. Wallace S. Foulds
Glasgow, Scotland

Ophthalmologists tend to regard the vitreous as a nuisance. Incarcerated in wounds it delays healing. It is implicated in the etiology of macular holes and retinal detachment. It is an ideal scaffold for cellular proliferation and is an excellent culture medium for bacterial growth.

It is no surprise that vitreo-retinal surgeons remove the vitreous given the least opportunity and can do so with apparent impunity and even benefit to the eye.

Given this background one may ask what advantage accrues from the presence of this highly structured ocular component.

In a word this may be homeostasis. Evidence for mechanical, hydrodynamic and biochemical homeostasis provided by the vitreous will be presented based on a variety of clinical and experimental findings including early attempts at pharmacologic vitreolysis, studies of fluid movement within the eye and clinico-pathological correlates.

NOTES:
VITREOUS STRUCTURE & PHARMACOLOGIC VITREOLYSIS

J. Sebag, MD, FACS, FRCOphth
VMRI Institute
Huntington Beach, California

Vitreous is a viscoelastic extracellular matrix that normally exists in a gel state as a result of the intricate organization of its macromolecular components. Hyaluronan (HA) and collagen, primarily type II but also type IX, and a hybrid of types V/XI, are organized into a three-dimensional network that maintains media clarity and provides shock-absorption. The peripheral vitreous cortex consists of densely packed collagen fibrils and has a high concentration of HA. In youth the posterior vitreous cortex is firmly adherent to the internal limiting lamina (ILL) of the retina. While the exact nature of vitreoretinal adhesion is not known, it most probably results from the biophysical properties of the extracellular matrix molecules found at this interface.

During aging there is weakening of vitreo-retinal adhesion and liquefaction of the gel, due to effects of transmitted light as well as the action of proteolytic enzymes and endogenous metalloproteinases. Concurrent weakening of the vitreo-retinal interface and liquefaction of the gel results in posterior vitreous detachment (PVD). Liquefaction without concurrent vitreo-retinal dehiscence results in Anomalous PVD, with traction exerted upon the retina or disruption of the posterior vitreous cortex with vitreoschisis. In the periphery, tears and detachments result while at the macula there can be vitreo-macular traction syndrome, macular holes, or premacular membranes with pucker. Thus, it would be desirable to alter vitreous and prevent Anomalous PVD.

Attempts to manipulate the gel state of vitreous and its attachment to the retina, known as Pharmacologic Vitreolysis, first began with hyaluronidase in 1949 and collagenase in 1973. A purified preparation of the former agent has recently failed to obtain FDA approval for clearing of vitreous hemorrhage without vitrectomy. Whereas this agent has also being proposed as a means by which to induce PVD, there is little theoretical basis for this approach, as hyaluronidase alone will not likely degrade the molecules responsible for vitreo-retinal adhesion. Indeed, experimental trials of hyaluronidase in rabbits failed to achieve PVD. On the other hand, combining hyaluronidase with CF has purportedly induced PVD in the rabbit, a finding also reported for plasmin combined with SF. Early observations of the effects of blood on vitreous laid the groundwork for approaches based upon extracting active agents from blood for pharmacologic vitreolysis.
Autologous plasmin was shown to induce PVD in rabbits and has since been used intraoperatively by Trese and Williams to facilitate pediatric and adult vitreo-retinal surgery.

Kroll and Gandorfer have also worked with this approach. Chondroitinase has been shown to disinsert vitreous from retina and facilitate surgical removal of premacular membranes. Because of the important role played by sulfated chondroitins in maintaining gel vitreous as well as mediating vitreoretinal adhesion, chondroitinasases hold great promise for pharmacologic vitreolysis. Dispase has proteolytic activity against type IV collagen and fibronectin, so it would seem well-suited as an agent to induce vitreo-retinal dehiscence, but would not likely liquefy vitreous. Indeed, studies by Del Priore & Kaplan found that dispase disrupted collagen fibrils within the lamina rara externa of the ILL of the retina with lesser effects upon the lamina densa. Those investigators concluded that these effects should not be deleterious to retinal function, since the foot plates of Mueller cells were intact. Their predictions were correct, as McCuen’s group found no abnormalities on electroretinography or any of the histologic and ultrastructural investigations that were undertaken after using dispase during vitrectomy in young pigs. It is important to note that in these two studies there was no evidence of vitreous liquefaction.

This underscores the concept that no single agent available today is likely to achieve both of the desired components of pharmacologic vitreolysis; i.e., liquefaction of the gel and vitreoretinal dehiscence. Dispase causes dehiscence but not liquefaction. Hyaluronidase liquefies vitreous gel but without gas probably does not induce PVD. Chondroitinase may do both but the depolymerization of HA and chondroitin sulfate only results in a reduction of vitreous gel wet weight and not gel destruction. Collagenases would probably need to be added to achieve such effects. Indeed, vitreous molecular morphology is so complex and there are so many different changes that occur with aging and various diseases, that the future will probably see the use of a mixture of agents whose relative concentrations will need to be adjusted depending upon the patient’s age, disease, and the desired effect; ideally beginning by inducing vitreoretinal dehiscence, followed by liquefaction of the gel vitreous.

NOTES:
MICROPLASMIN INDUCED VITREOLYSIS AND POSTERIOR VITREOUS SEPARATION IN PORCINE EYES

Prof. Marc de Smet
Department of Ophthalmology,
University of Amsterdam
Department of Morphology,
Netherlands Ophthalmic Research Institute Amsterdam,
The Netherlands

Purpose:
Microplasmin is a low molecular weight recombinant protein containing the catalytic domain of human plasmin. Its catalytic activity is similar to full length plasmin. Initial experiments in porcine eyes were carried out to determine its potential in inducing posterior vitreous separation, and vitreolysis.

Method:
Microplasmin at different concentrations was injected via the pars plana in pig eyes immediately following death at escalating concentrations from 0.0625 mg to 0.39mg. Bovine plasmin was used as a control at a concentration of 2 U in 0.1 mL At time 0, 15, 30, 60, and 120 minutes after injection, the eyes were fixed in Peter’s solution and placed on ice to stop further enzymatic activity. Eyes were carefully dehydrated prior to processing to minimize disturbance to the intraocular architecture. Eyes were examined by conventional microscopy and scanning electron microscopy.

Results:
Vitreo-retinal adhesions were markedly diminished in treated eyes out to the ora serrata, particularly at concentrations equal or above 0.125 mg. There was also significant change to the fibrillar structure of the vitreous, suggesting vitreolysis.

Conclusion:
A recombinant protein – microplasmin appears to possess many of the enzymatic characteristics of plasmin. Further investigations may determine its potential for clinical use.

NOTES:
ANATOMICAL STRUCTURES IN THE PERIPHERAL VITREORETINAL INTERFACE

Stephan Dunker, MD
Troisdorf, Germany

The ultrastructural nature of the preequatorial vitreoretinal interface was studied to elucidate the predisposing role for peripheral retinal tear formation.

Fourteen enucleated globes from seven deceased patients were examined. The patients ranged in age from 16-88 years, with an average age of 40 years. None of the patients had a history of ocular or systemic disease that could have affected the eyes. Globes were examined by stereomicroscopy and by light and electron microscopy using the celloidin embedding method.

On examination there are several structures in addition to tufts and rosettes. In the vitreous cortex, fibrillar structures with no vitreoretinal attachment to the retina frequently were found. These structures could be called "tubuli" because of their spiral appearance. Mushroom-like structures, which could be referred to as "spiculae," were found to arise from the intact internal limiting lamina of the retina and inserted into the vitreous cortex, constituting foci of vitreoretinal adhesion. Other structures, which could be called "verrucae," arose from the disrupted internal limiting lamina of the retina and inserted into disrupted areas of vitreous cortex.

Tubuli appear to be remnants of the embryonic vasculature with no clinical or pathologic significance. Because of their pattern of inserting into the internal limiting lamina of the retina and the peripheral vitreous cortex, spiculae and verrucae may play an important role in the formation of retinal breaks.

NOTES:
AGING OF THE VITREOUS BASE

Paul Bishop PhD, FRCS, FRCOphth
Wellcome Trust Senior Research Fellow in Clinical Science
Honorary Consultant Ophthalmologist
University of Manchester
Manchester, England

Purpose:
To determine the width of the posterior vitreous base in human eyes of different ages and to clarify the nature of the postoral retinovitreal adhesion.

Methods:
The posterior limit of the vitreous base was delineated after mechanical peeling of the post-basal vitreous cortex from the retina in 58 pairs of eye bank eyes, and the area of residual retinovitreal adhesion was measured. Scanning electron microscopy was performed on the undersurface (or retinal aspect) of the inner limiting lamina (ILL) after trypsin digestion of the peripheral retina.

Results:
An age-dependent increase in the width of the posterior vitreous base was revealed which was greater in male vs. female donors and in the nasal half vs. the temporal half of the globe. Ultrastructural studies revealed progressive invasion of the innermost peripheral retina by collagen fibrils that eventually formed a dense sublaminar matting in the elderly. The collagen fibrils penetrated the ILL through localised defects and intertwined with those in the basal gel.

Conclusions:
With ageing, the posterior border of the vitreous base migrates posteriorly. Intraretinal synthesis of collagen fibrils, their penetration of the ILL and their splicing with cortical vitreous fibrils, underlie the slowly evolving retinovitreal adhesion.

NOTES:
CHARACTERIZATION AND HISTOGENESIS OF THE INTERNAL LIMITING LAMINA (ILL)

Andrew W. Eller, MD and Willi Halfter, PhD
Department of Ophthalmology and Department of Neurobiology,
University of Pittsburgh School of Medicine
Pittsburgh, PA USA

Introduction:
Basal laminae form a physical barrier between epithelia and the underlying connective tissue. The retinal basal lamina, also referred to as the Internal Limiting Lamina (ILL), separates the retinal neuroepithelium from the vitreous. Recent in vivo experiments have shown that the retinal and pial basal laminae are particularly important during the development of the nervous system by organizing and maintaining the cytoarchitecture of the pial and vitreal surface of the brain and retinal neuroepithelium, thereby having a crucial function during axonal navigation and histogenesis.

Methods:
Immunocytochemical staining of cross-sections of paraformaldehyse-fixed eyes from chicks was performed. To determine the site of synthesis of the retinal basal lamina proteins, in situ hybridization studies were done on cross-sections of E5, E6, and E10 eyes using cRNA probes to nidogen, collagen XVIII, agrin, the 1-chain of collagen IV, and perlecan. Experiments were also performed to determine the role of vitreous in ILL formation and whether the ILL regenerates in vivo.

Results:
The major constituents of the retinal basal lamina are laminin, nidogen, agrin, and collagen XVIII. Collagen IV was detectable after preincubation with pronase. Collagen XVIII mRNA was detected in the future ciliary body and the optic disc, and was undetectable in the neural retina all stages of eye development studied. Nidogen mRNA was also detected in ciliary body, and optic disc. It was abundantly expressed by the lens epithelial cells, and not, or in only minor quantities, in the neural retina. Agrin mRNA was detected in ganglion cells of the retina, and was the only basal lamina protein that was synthesized by the neural retina. Perlecan and 1-chain of Collagen IV were detected in the lens (but not ciliary body or retina). Abundant 1 collagen IV mRNA was found in the optic disc, in the optic nerve, and in the periocular connective tissue.
Experimentally, the basal lamina does not form over inverted retinal transplants when the vitreous has been removed. On the other hand, it does form when only the lens has been removed.

**Conclusions:**
Almost of the retinal basal lamina proteins are synthesized in extraretinal tissues of the eye, such as the ciliary body, the optic disc, and lens epithelium. The mRNA distribution pattern is unique and different basal lamina proteins have different tissue sources. Intravitreal and intraretinal blood vessels are additional sites of synthesis for basal lamina proteins in the mouse eye. The vitreous body represents a storage compartment for basal lamina constituents that allows for the continuous and instantaneous assembly of the retinal basal lamina. The retinal neuroepithelium provides appropriate receptors for the immobilization of one or several basal lamina proteins, which then serve as an anchor for other proteins to associate to complete the basal lamina. Candidate receptor proteins for basal lamina nucleation are the members of the integrin family and the laminin-binding dystroglycan. Dystroglycan is specifically localized to the neuroepithelial endfeet at the vitreal and pial border next to the retinal and pial basal lamina.
VITREO—RETINAL SURGERY I

Moderators: Carl Claes, MD & Thierry Verstraeten, MD

Unoperated Eyes with Persistent Fetal Vasculature ............... 13:30-13:50
William Tasman, MD

Posterior Zone Retinopathy of Prematurity ......................... 13:50-14:10
Tatsuo Hirose, MD

Are Prior Retinopexies an Influential Factor in the Development of Macular Hole
Alvaro Rodriguez, MD

BREAK/EXHIBITS OPEN ........................................... 14:30-15:15

VITREO—RETINAL SURGERY II

Moderators: Thierry Verstraeten, MD and Carl Claes, MD

Pharmacotherapy for PVR ........................................... 15:15-15:45
David Charteris, MD

PVR Surgery ......................................................... 15:45-16:15
Carl Claes, MD

Innovations in Vitreo-Retinal Surgery ............................. 16:15-16:45
Eugene de Juan, MD

Macular Hole Surgery: A Comparison of ILL Peeling .............. 16:45-17:00
With and Without ICG
John Gonder, MD

Fluorescence of Optic Nerve and Macula after ................... 17:00-17:15
ICG Assisted Macular Surgery
Laurent Francois, MD

GALA DINNER .............................................................. 19:00-21:00
Belfry Tower
UNOPERATED EYES WITH PERSISTENT FETAL VASCULATURE (PFV)

William Tasman, M.D.
Wills Eye Hospital
Philadelphia, PA

Purpose:
To present a follow-up on children with PFV when surgery was not performed.

Methods:
From January 1992 through June 2001, 31 infants (31 eyes) with PFV were evaluated to document the number of operated eyes versus unoperated eyes to see whether the latter group remained stationary or became worse.

Results:
Seventeen of the 31 eyes underwent surgery. Of the fourteen unoperated eyes seven were judged to be inoperable. The remaining seven eyes were not operated because the fundus could be visualized through an undilated pupil. Follow-up on 5 of the 7 eyes ranged from 18 months to 9.5 years. None showed progression of the lens opacity or development of retinal detachment. The eyes were amblyopic, but functional.

Conclusions:
In this limited series of eyes with PFV where the plaque was not large enough to fill the pupil, the eyes on follow-up were amblyopic, but useful vision was retained and the lens opacity did not enlarge.

Dr. Nishi Gulati assisted in this study.

NOTES:
POSTERIOR ZONE RETINOPATHY OF PREMATURITY

Tatsuo Hirose, M.D.
Schepens Retina Associates
Boston, MASS, USA

Zone 1 and posterior Zone 2 disease (Posterior Zone) are probably the most severe types of Retinopathy of Prematurity. The flat extraretinal neovascularization may develop on the surface of the vascularized retina particularly on the temporal side of the fundus without going through stages 1 and 2. This flat neovascularization may be easily missed. Fluorescein angiography shows profuse leakage from the flat neovascularization. Laser ablation on the avascular retina is effective in shrinking the neovascularization. Retinal detachment associated with no neovascular activities with no plus disease may be dealt with closed vitrectomy with or without lensectomy or open sky vitrectomy depending upon the clinical presentations of the disease in each case.

The author describes the clinical features in 34 cases with posterior zone ROP including fluorescein angiography, the results of laser treatment and vitrectomy, both closed and open sky, for totally detached retina. Prevention of retinal detachment is most important for these infants to develop vision. However, some eyes with totally detached retina can still be saved by vitrectomy.

NOTES:
ARE PRIOR RETINOPEXIES AN INFLUENTIAL FACTOR IN THE DEVELOPMENT OF MACULAR HOLES?

Alvaro Rodríguez, MD
Bogotá—Colombia

Introduction:
Macular holes have been observed after retinopexies which are believed to be due to biologic tractional / mechanical alterations at the vitreoretinal interface.

Methods:

Results:
Twenty-two eyes of twenty patients (45% older than 60 years, 60% myopic, PVD 81%, and cataract in 55%) were studied. On follow-up, of 91% peripheral retinal breaks (41% alone and 59% with retinal detachment), 50% developed premacular membranes and 100% macular holes, 50% of early onset, late in 45%.

Initial management of peripheral breaks was laser / cryocoagulation; scleral buckling in 59% eyes with retinal detachment; 3 of 4 recent macular holes were closed with vitrectomy; 17 eyes were not treated -before 1991. Follow-up indicated that 10% improved, 36% were stable, and 54% worsened.

Conclusions:
Macular holes may develop after retinopexies. The main risk factor is surgical trauma influencing contracting epiretinal membranes. Limitations of retrospective methods require prospective studies. However, the clinical profile of cases suggest retinopexies as an influential factor, further suggesting the avoidance of extensive prophylactic retinal surgery and improvements in coagulation technology.

NOTES:
PHARMACOTHERAPY OF PROLIFERATIVE VITEORETINOPATHY

David G. Charteris MD, FRCS, FRCOphth
Vitreoretinal Unit, Moorfields Eye Hospital
London, UK

Proliferative vitreoretinopathy (PVR) has been seen as a target for adjunctive treatment for many years. PVR remains a significant complication in primary retinal detachment surgery, recent series documenting incidences of between 5 and 12%. Additionally it is a major problem in ocular trauma, complex forms of retinal detachment such as giant retinal tears and is an important complication of retinal rotation/translocation for AMD. Adjunctive medication which allowed vitreoretinal surgeons to reduce or eliminate the fibrocellular response associated with PVR (together with an ability to predict “high-risk” cases) would be of immense value.

Initial uncontrolled clinical studies on adjunctive agents in retinal surgery produced promising results and importantly the intraocular use of the various agents did not appear to produce any toxicity. Laboratory studies were also promising but may have been compromised by the use of inadequate experimental models.

The first randomised controlled trial of adjuncts in PVR surgery was conducted by a group of European centres to investigate the potential of daunorubicin to improve success rates in surgery for established PVR. A ten minute infusion of daunorubicin (7.5µg/ml) was given during vitreoretinal surgery after membrane peeling and retinectomy. The trial demonstrated a significant reduction in the rate of re-operations and the primary outcome measure (retinal reattachment without further surgery at 6 months) showed a trend towards improvement which narrowly failed to reach significance (p=0.07). Nevertheless this is the first controlled clinical trial to demonstrate a pharmacological effect on the natural course of PVR.

A group of researchers in the United Kingdom have conducted two clinical trials on the use of the combination of intraocular 5-fluorouracil (5FU) and low molecular weight heparin (LMWH) in cases at high risk of PVR and those undergoing surgery for established PVR.

To test the efficacy of a combination of these agents delivered at the time of surgery, “high-risk” cases of retinal detachment undergoing primary vitrectomy and gas tamponade were selected on the basis of clinical characteristics, using a formula derived from regression analysis.
Cases were then randomised to have infusion fluid with or without the addition of 5FU 200µg/ml and LMWH 5IU/ml for one hour (the infusion was changed if surgery continued beyond one hour). The combined adjunctive treatment significantly reduced the incidence of post-operative PVR (from 26.4% to 12.6%). No adverse treatment-related events were identified in the study. A further randomised trial of the same adjunct combination on patients undergoing vitrectomy and silicone oil exchange for established PVR (grade C or more) did not appear to show a beneficial effect of treatment.

peripheral retinal breaks (41% alone and 59% with retinal detachment), 50% developed premacular mem

References:


PVR SURGERY

Carl Claes, MD
Antwerp, Belgium

NOTES:
INNOVATIONS IN VITREORETINAL SURGERY

Eugene deJuan, Jr., MD
Doheny Retina Institute
Keck/USC School of Medicine
Los Angeles, CA

Over the past few years there has been an explosion of new surgical treatments for vitreoretinal diseases. These include innovations such as transconjunctival 25 gauge vitreous surgery, minimally invasive sheathotomy, central vein cannulaization, new developments in retinal electronic prosthetic devices and a variety of new drug delivery devices.

We will review the instrumentation and results of the 25 gauge system and review our experience with several new surgical methods.

NOTES:
MACULAR HOLE SURGERY: A COMPARISON OF INTERNAL LIMITING LAMINA PEELING WITH AND WITHOUT ICG

John Gonder, MD
Ivey Eye Institute
London, Ontario, Canada

A retrospective study of 173 consecutive eyes that underwent macular hole surgery was performed comparing the surgical outcomes of the eyes with ICG-assisted ILL peeling to those eyes with ILL peeling. The two groups were comparable in terms of macular hole duration, stage and preoperative visual acuity.

Failure of macular hole closure occurred in 2.5% of the ICG group versus 20% in the no ICG group (p<0.01). Re-opening of the macular hole occurred in 9.5% of the no ICG group and in no patients in the ICG group (p<0.01). The post-operative Snellen visual acuities were similar between the two groups.

ICG assisted ILL peeling improved macular hole closure and reduced macular hole re-opening. The use of ICG dye did not appear to have any significant detrimental effect on post-operative visual acuity.

NOTES:
FLUORESCENCE OF THE OPTIC NERVE AND THE MACULA AFTER STAINING WITH INDOCYANINE GREEN FOR INTERNAL LIMITING LAMINA PEELING IN MACULAR DISEASES

Laurent Francois, MD
Bordeaux, France

Purpose:
Indocyanine green (ICG) can be injected in the vitreous cavity after vitrectomy to stain the internal limiting lamina (ILL). This staining facilitates the peeling of the ILL in the macular area. Toxicity of ICG has been described. Persistent staining of the disc has recently been published (R. Spaide, Retina 2002). The purpose of our study is to evaluate the long term infrared fluorescence of the macula and the optic nerve after vitrectomy and staining with ICG of the ILL in cases of macular diseases.

Methods:
A consecutive series of 19 eyes of 18 patients with macular hole (14 eyes), branch vein occlusion (3 eyes), macular edema (1 eye) and macular pucker (1 eye) was studied in a prospective manner. During surgery, after vitrectomy, 0.1cc of ICG (Infracyanine® SERB) was injected over the posterior pole, left in place during 1 minute and removed. The ILL was then easily removed from the macular area. After surgery, digital fundus infrared photographs were taken with the excitation and blocking filters used for ICG angiography during follow up.

Results:
Mean follow up was 10.6 months (2.6-18.4 months). We observed infrared fluorescence of the macula in 14 eyes, of the optic nerve in 17 eyes. Of the 10 eyes with at least 1 year follow up, 8 demonstrated a less intense but persistent infrared fluorescence of the optic nerve, and 6 an infrared fluorescence of the macula. No other change was observed in the fundus that could be related to a possible toxic effect of the dye.

Conclusions:
ICG staining facilitates the removal of the ILL but persistent infrared fluorescence of the macula and the optic nerve can be observed over 1 year after surgery. The safety of ICG has to be confirmed by further studies.
ARMD PATHOGENESIS & CLINICAL DIAGNOSTICS

Moderators: Prof. G. Coscas, MD and Prof. G. Soubrane, MD

Pathogenesis of Age-Related Macular Degeneration ............... 8:00-8:30
  Gregory Hageman, PhD

Evolution of Early ARMD Correlates with Basal Deposits .......... 8:30-8:50
  Shirley and John Sarks, PhD

ICG Angiography Classification in ARMD. ....................... 8:50-9:20
  Prof. Gabriel Coscos

Does a Macula-Off Retinal Detachment Influence the Pathogenesis of AMD? A Long Term Follow-up
  Prof. Peter Kroll

BREAK/EXHIBITS OPEN ............... 9:40-10:10

PAUL KAISER AWARD I

Novel Treatment Strategies for AMD ....................... 10:10-11:10
  Eugene de Juan, Jr., MD

ARMD THERAPEUTICS

Moderators: Prof. Gisele Soubrane and Prof. Gabriel Coscas

Anti-VEGF Therapy in Exudative ARMD ..................... 11:10-11:40
  Prof. Gisele Soubrane

RPE Transplantation in Advanced ARMD ..................... 11:40-12:10
  Prof. Susanne Binder

Management of Submacular Hemorrhage ..................... 12:10-12:30
  Prof. Marc de Smet

Transpupillary Thermotherapy of Occult Choroidal Neovascularization ..................... 12:30-12:45
  Prof. Marie Benedicte Rougier

LUNCH/EXHIBITS OPEN ..................... 12:45-14:15
EVIDENCE FOR THE PARTICIPATION OF INFLAMMATION AND IMMUNE-MEDIATED PATHWAYS IN THE ETIOLOGY OF AGE-RELATED MACULAR DEGENERATION

Gregory S. Hageman, Ph.D.
Center for Macular Degeneration,
Department of Ophthalmology and Visual Science University of Iowa

Age-related macular degeneration (AMD), the leading cause of irreversible blindness in the developed world, affects approximately 15% of individuals over the age of 60. A number of population-based studies indicate that this disease is heritable as an autosomal dominant trait in a significant portion of afflicted individuals, although a disease-causing gene(s) has not yet been identified. AMD is characterized by the progressive loss of central vision that is attributable to the deposition of extracellular drusen and basal laminar deposits, often followed by atrophic, exudative and/or hemorrhagic changes within the macula.

The sequelae of cellular and molecular events that are associated with these changes are poorly understood. A new paradigm is emerging, however, that may help to explain the etiology of AMD. We and others have generated multiple lines of evidence that implicate a major role for local inflammatory and/or immune-mediated processes at the level of the ocular retinal pigmented epithelium-choroid interface in the development of drusen and the pathobiology of AMD. Our studies have also led to the observation that dendritic cells, potent antigen-presenting cells, are intimately associated with drusen development and that complement activation is a key pathway that is active both within drusen and along the RPE-choroid interface. Moreover, we have generated evidence that these pathways may be directly or indirectly related to other systemic and/or extraocular diseases, including Alzheimer’s disease and atherosclerosis.

In the severest form of AMD, choroidal neovascular membranes breech the macular Bruch’s membrane and gain access to the sub-RPE and subretinal spaces. Interestingly, the biological pathways that are associated with the predilection of the macula toward atrophic and exudative degeneration have not been elucidated. Recent morphometrical analyses conducted in our laboratory have revealed a statistically significantly difference in the integrity (p<0.00001) and thickness (p<0.00001) of the elastic lamina between the macula and extramacular regions of donors.
and at all ages. Donors with a clinical history of AMD have a significantly lower mean integrity in the macula, as compared to that of age-matched donors without AMD (p=0.0015). We propose that these differences may help to explain why the macula is more susceptible to degenerative changes in AMD.

A working model of AMD pathobiology that encompasses these findings has been advanced. Although clearly a paradigm shift in our understanding of the biological processes involved in AMD, the downstream consequences of active, immune-mediated pathways appear to induce significant damage to macular cells and tissues, contributing to choroidal neovascularization and severe vision loss.

NOTES:
EVOLUTION OF EARLY AGE-RELATED MACULAR DEGENERATION: CORRELATION WITH BASAL DEPOSITS

Shirley Sarks, PhD and John Sarks, PhD
Medical Research Institute, Randwick
Sydney, Australia

PURPOSE:
To examine the relationship of basal laminar deposit and membranous debris (basal linear deposit) to the evolution of early AMD.

METHODS:
530 eyes representing normal aging or early AMD and previously submitted to clinicopathological examination were graded for (1) amount and type of basal laminar deposit (BLD), and (2) quantity of membranous debris. The latter is not seen in histological sections but a means of estimating its presence is described.

RESULTS:
Early-type (banded) BLD and membranous debris appear initially in normal aged eyes. In AMD the preferential accumulation of debris internal or external to the basement membrane of the retinal pigment epithelium (RPE) appears to determine whether an eye presents with pigment changes or soft drusen. When RPE/photoreceptor fallout exceeds a certain threshold membrane production declines while late (amorphous) BLD production increases.

CONCLUSIONS:
Normal aging and AMD form a continuum. The quantity of membranous debris and type of BLD mirror the state of the RPE/photoreceptors.

NOTES:
COULD ICG ANGIOGRAPHY IMPROVE DETECTION AND PROVIDE USEFUL CLASSIFICATION OF CNV IN AMD AND FOR FOLLOW UP POST TREATMENT?

Prof. Gabriel Coscas, Prof. Gisèle Soubrane
Clinique Ophtalmologique Universitaire de Créteil, Paris XII

Purpose:
Fluorescein angiography (FA) was the “Gold standard” for the assessment of macular neovascularization and classification. But infrared angiography with infracyanin green (ICG-A) could allow better visualization of new vessels that are choroidal in origin.

Material and Method:
Prospective study on consecutive patients presenting with exudative AMD in the department, during one month, analysing the data given by infracyanin-green video-angiography with SLO-HRA from Heidelberg with confrontation with the results of digitalized FA for detection classification and follow up post-treatment of CNV in AMD.

Results:
At early stage of Age Related Maculopathy, detection and localization of soft drusen was easy with ICG-A, in all cases allowing analysis of extension, confluence and/or association with serous RPE detachment that are uniformly hypofluorescent in ICG-A.
ICG Angiography was useful for detection and as confirmation-test when FA showed localised area of hyperfluorescence due to early development of CNV, associated with many drusen and RPE changes.
ICG Angiography was useful for precise analysis of the different components of the lesion, not only perfusion and area of CNV but also associated fibrosis and/or atrophy.
Moreover, ICG Angiography was able to analyse the exact topography and extension of the area of the CNV in relation to the center of the fovea, to detect their perfusion and the presence of one or many feeder vessels.

Choroidal new vessels could be separated into “active CNV” (with early perfusion and late wash-out) and “slow CNV” (with late perfusion and progressive coloration and - for a few of them - very late leakage).
Most of the so-called “occult CNV in FA” were converted into well-defined CNV with precise delimitation of their borders. Nevertheless, a group of them were detectable only on late stage as a hyperfluorescent plaque, with more or less defined borders.

During follow-up after treatment, ICG-A was critical for the early detection of persistance/recurrence status-post juxta-foveal thermal laser (where FA was not as effective). ICG-A was also useful for the analysis of perfusion of residual (or recurrent) CNV after PDT with Verteporphin and to help with indications of re-treatment.

**Conclusions:**
ICG-Angiography with SLO-HRA appeared to be useful and efficient to recognize CNV in AMD that are active and need immediate treatment -as they are perfused early with late wash-out; ICG-A is also efficient to analyze the perfusion, localization and extent of CNV, even if they are “occult” in FA or masked in a PED or by a thin layer of blood; and then to guide the treatment or re-treatment with thermal laser or with PDT.

ICG-A with SLO-HRA should be recommended as a very useful guide for the management of CNV in AMD.

**NOTES:**
DOES PREVIOUS RETINAL DETACHMENT AFFECT THE INCIDENCE OF NON-VASCULAR AND VASCULAR AMD?

Prof. Peter Kroll  
Department of Ophthalmology  
Phillips-Universitat

Background:  
After retinal detachment (RD) there are changes in the retinal pigment epithelium (RPE) with possible effects on age-related macular degeneration (AMD). We evaluated the incidence of non-vascular and vascular AMD in patients aged 60 years and older with previous RD.

Methods:  
We evaluated 66 patients after scleral buckle for a rhegmatogenous RD including the macula who had a follow-up of >5 years.

Results:  
All treated eyes demonstrated greater RPE alterations compared to the fellow eye. 14 out of 66 patients (21.2%) developed uni- or bilateral signs of AMD. Nonvascular AMD was seen in 3 treated and 7 untreated eyes. Vascular AMD were present in 8 treated, whereas 3 fellow eyes 7 out of the 8 treated patients with vascular AMD demonstrated a pigment epithelial detachment (PED). CNV developed in 1 treated and 3 untreated fellow eyes.

Conclusion:  
RD alters the physiology of the RPE. Patients with AMD have a lower incidence of CNV after RD. The exact etiology and mechanism(s) are presently unknown.

NOTES:
Age-related Macular Degeneration is increasingly a cause of visual loss in developed nations. While the precise molecular mechanisms are becoming clearer, the options for treatment remain limited in both number and effect. At the Doheny Retina Institute we have developed a structure that allows rapid development of potential new therapeutics by combining early pre-clinical efficacy and early stage clinical testing to demonstrate “proof of concept”.

I will present three projects under study at the institute for the treatment of AMD. The first is a pilot study of sub-retinal steroid injection. In this IRB-approved study we have enrolled 8 patients with 4 months average follow up. 3 of the eight patients have >2 lines of visual improvement. The second study is the use of oral retinoids in the treatment of occult CNV. In this IRB-approved study currently 3 of 7 patients have improved. The final study is the use of oral isoflavone for the treatment of CNV. New methods of drug delivery will also be presented.
ANTI-VEGF THERAPY IN EXUDATIVE ARMD

Prof. Gisele Soubrane
Creteil, France

NOTES:
Abstract

TRANSPLANTATION OF AUTOLOGOUS RETINAL PIGMENT EPITHELIUM CELLS

Prof. Susanne Binder
Department of Ophthalmology,
Rudolf Foundation Clinic
Vienna, Austria

We present our two year results with transplantation of autologous retinal pigment epithelium cells (RPE) in eyes with foveal choroidal neovascularisation related to age related macular degeneration. Those eyes where transplantation of RPE was not possible served as controls. Examinations included best corrected visual acuity for far and near, biomicroscopy, visual fields, fluorescein and indocyanin-green angiography, multifocal ERGs and optical coherence tomography at 3 months intervals up to 2 years.

Statistical analysis of distance visual acuity showed significant improvement after 3, 6, 9, 12 and 15 months in the transplantation group and no significant difference in the control group. We conclude that transplantation of autologous RPE might be considered as a treatment option for patients with exudative Age-related Macular Degeneration.

Other Authors:
Ilse Krebs, Ulrike Stolba, Ali Abri, Christian Jahn, Hans Feichtinger, Lukas Kellner, Adele Assadoullina, Prof. Dr. Hilgers, Boris Stanzl

NOTES:
MANAGEMENT OF SUBRETINAL HEMORRHAGE

Prof. Marc D. de Smet
Department of Ophthalmology,
University of Amsterdam

The management of subretinal, in particular submacular, hemorrhage has greatly evolved over the last decade. A wide variety of options both surgical and medical have been developed, all with the aim of clearing blood from the central visual axis so as to minimize permanent damage to macular function. Operating room procedures that were advocated early on have been relegated to large subretinal hemorrhages in select group of monocular patients. Medical approaches using gas tamponade for 12 to 72 hours with or without the use of TPA are currently preferred. The need to address and treat the underlying cause once blood has been displaced should also be stressed as an essential element of any treatment plan or protocol. The approach used in the University of Amsterdam will be discussed in the light of current literature.

NOTES:
TRANPUPILLARY THERMOTHERAPY OF OCCULT CHOROIDAL NEOVASCULARIZATION

Prof. Marie Benedicte Rougier
University Hospital,
Bordeaux, France

Purpose:
To assess the efficacy of transpupillary thermotherapy (TTT) for the treatment of occult choroidal neovascularization (CNV).

Methods:
A retrospective, non-randomized, study of 66 eyes of 66 patients with occult CNV in age-related macular degeneration (AMD). Initial ETDRS visual acuity ranged from 20/200 to 20/40. TTT was applied with a 810nm-diode laser (BVI, Quantel). Two independent physicians evaluated the results through visual acuity, fundus and angiography examination, prior to treatment and at one year. A visual acuity change of 3 lines in the ETDRS chart was considered clinically significant.

Results:
9 patients were excluded. Among the remaining 57 patients, visual acuity remained unchanged only in 10%, when 22 patients presented severe complications, as atrophic retinal scar (3) or occurrence of classic new vessels (19).

Conclusions:
As we have disappointing results, we have stopped treating occult CNV with TTT. However, this technique could be reconsidered if we can adjust the parameters of the laser in accordance with retina pigmentation and lens opacities.

NOTES:
**MEDICAL RETINA/UVEITIS**

*Moderator: Jean Jacques DeLaey, MD*

- **Genotype/Phenotype Correlation: Myth or Reality** 14:15-14:45  
  Jean Jacques DeLaey, MD

- **The Prospect of Biological Treatment of Retinal Dystrophies** 14:45-15:15  
  Prof. Alan Bird

- **Pathogenesis of Uveoretinitis: Lessons from EAU** 15:15-15:45  
  Janet Liversidge, MD *(for Prof. John Forrester)*

- **Transplantation of Glial Progenitor and Neural Stem Cells Into The Normal and Dystrophic Retina** 15:45-16:05  
  Prof. Gisbert Richard

- **Occult Macular Dystrophy** 16:05-16:25  
  Yozo Miyake, MD

- **DHA Supplementation in X-linked RP** 16:25-16:45  
  Rand Spencer, MD

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**ADJOURNMENT** 16:45

**SPECIAL AWARD PRESENTATION TO** 17:00  
**DR. CHARLES L. SCHEPENS**

**DINNER AT OLD ST. JOHN HOSPITAL** 19:00-22:00
GENOTYPE/PHENOTYPE CORRELATIONS IN RETINAL DYSTROPHIES: MYTH OR REALITY?

J.J. De Laey, MD
Department of Ophthalmology,
Ghent University Hospital
Belgium

Background:
Genotype-phenotype correlation studies will become more and more important for the understanding of pathogenic mechanisms in retinal dystrophies.

Methods:
To illustrate the difficulties of phenotype-genotype correlation, two groups of diseases will be discussed: Leber’s Congenital Amaurosis (LCA) and Peripherin/RDS related dystrophies.

Results:
Leber described the association of congenital blindness, nystagmus and a fundus aspect which may be variable but can be similar to that seen in retinitis pigmentosa. The disease has an autosomal recessive mode of inheritance. In 1954 Franceschetti and Dieterle noted absent or markedly reduced ERG as the 5th characteristic of this condition. To date, mutations in at least 6 genes have been associated with this condition and probably more, still unknown genes are involved in its pathogenesis. Although there may be differences in presentation with the different genes involved, LCA may be considered as an example of a similar phenotype related to different genotypes.

On the other hand mutations in the peripherin/RDS gene may be the cause of different clinical manifestations in a same family such as retinitis pigmentosa, fundus flavimaculatus and pattern dystrophy.

Conclusions:
These two examples illustrate the complexity of genotype-phenotype correlations and stress the necessity of an even more precise clinical diagnosis and of further genetic studies.

NOTES:
Abstract

THE PROSPECT OF BIOLOGICAL TREATMENT OF RETINAL DYSTROPHIES

Prof. Alan Bird
Moorfield’s Eye Hospital
London England

Three biological approaches to treatment of retinal dystrophies have been developed, namely the use of growth factors, gene therapy and cell transplantation. It is important for the clinician to prepare for the introduction of treatment into clinical practice, so that the potential benefits of scientific research can be realized.

Clinical Setting for Treatment:
It is evident that a large well-documented patient pool is necessary to realized the full benefits of treatment, should this be introduced into clinical practice. Certain attributes of disease should be established.

1. Identification of the causative genes: Currently it should be possible to identify the responsible gene in about 50% of families with autosomal dominant disease, and most if not all X-linked disease. For autosomal recessive disease the number is uncertain. To achieve this objective genotyping laboratories are required.

2. Knowledge of the disease mechanisms: Whether disease is due to haplo-insufficiency or gain of function will determine a suitable approach to gene therapy. Equally important is the identification of the cell that expresses the mutant gene.

3. Temporal profile of functional loss and cell death: It has been shown that in some disorders cell dysfunction may precede cell death by several years and in others loss of function is due to cell death. In the first case gene therapy may cause recovery of function, and cell transplantation would be unsuitable. In the second cell transplantation would be appropriate and gene therapy may slow the degenerative process.

4. Detection of the therapeutic effect: The techniques of recording the treatment effect must be available such as electroretinography, psychophysics and specialized imaging.

To achieve these objectives it will take a great deal of time and effort on the part of clinicians, scientists and patients.
Abstract

PATHOGENESIS OF UVEORETINITIS: LESSONS FROM EAU

Janet Liversidge, MD for Prof. John Forrester
Dept of Ophthalmology,
University of Aberdeen

Uveoretinitis, or posterior uveitis as it is more commonly known, is a major cause of sight threatening ocular disease. Experimental autoimmune uveoretinitis (EAU) is a good model for study of the pathogenesis of EAU. Despite the fact that the retina is an immune privileged site, inflammation can be induced. Breakdown of tolerance appears to be the main mechanism and likely involves the blood retinal barrier. Using a scanning laser ophthalmoscopy-based in vivo system, we have identified factors which regulate traffic of inflammatory cell across the blood retinal barrier. In addition, we have identified new potential ways to inhibit EAU using a dendritic cell based immunotherapy regime. These studies will be described in this presentation.

NOTES:
Abstract

TRANSPLANTATION OF GLIAL PROGENITOR AND NEURAL STEM CELLS INTO THE NORMAL AND DISEASED RETINA

Prof. G. Richard
University Eye Hospital of Hamburg
Germany

Purpose:
To evaluate the use of intraretinal transplantations of oligodendrocyte progenitor cells (OPCs) to study axon-oligodendrocyte interactions in vivo, and of intraretinal transplantations of neural stem cells (NSCs) and retinal stem cells (RSCs) to treat retinal dystrophies.

Methods:
OPCs were isolated from cerebral hemispheres of neonatal rats, NSCs from the spinal cord of embryonic mice, and RSCs from the ciliary margin of the adult mouse eye. OPCs were grafted into the retina of young rats, and NSCs were grafted into the dystrophic retina of adult β2/β1 knock-in mutant mice. RSCs were characterized in vitro.

Results:
Numerous oligodendrocytes were present in the nerve fiber layer of host retinas one month after transplantation of OPCs. Donor-derived oligodendrocytes myelinated a large portion of the nerve fiber layer of experimental retinas, and formed ultrastructurally intact myelin around ganglion cell axons. NSCs integrated extensively into retinas of adult β2/β1 knock-in mice and populated all retinal layers, including the outer nuclear layer. NSCs survived for extended periods of time in host retinas, and differentiated into astrocytes and myelin-forming oligodendrocytes, but not into nerve cells. Cells with properties characteristic of RSCs were isolated from the ciliary margin of the adult mouse eye.

Conclusions:
Extensive myelination of intraretinal segments of ganglion cell axons after transplantation of OPCs demonstrates the competence of these axons to become myelinated along their entire length. We thus conclude that non-neuronal factors at the retinal end of the optic nerve prevent migration of OPCs from the nerve into the retina, and as a consequence intraretinal myelination. Intraretinally grafted NSCs integrate into the dystrophic retina of adult mice and differentiate into neural cell types, but fail to differentiate into retinal cell types. Cell therapy of dystrophic retinas thus requires the availability of stem cells displaying the ability to differentiate into retina-specific cell types. Pigmented cells from the ciliary margin of the adult mammalian eye show characteristics of stem cells, and differentiate into retina-specific glial and neuronal cell types in vitro.
OCCULT MACULAR DYSTROPHY

Yozo Miyake, MD and Hiroko Terasaki, MD
Department of Ophthalmology, Nagoya University

Occult macular dystrophy (OMD) is a newly identified clinical entity with autosomal dominant inheritance (Miyake et al: AJO 1990, 1996 IOVS 1999). The clinical findings are progressive decline of visual acuity, color vision deficiency, normal fundus, normal fluorescein angiography, normal full-field ERG, and abnormal focal macular ERG (FERG). We now summarize 40 patients with OMD to identify common functional abnormalities.

The ages of these 40 patients (24 male and 16 female) ranged from 8 to 70 years. Two or more affected members were detected in 7 pedigrees, of which 6 families are suggested to show autosomal dominant inheritance. Fundus, fluorescein angiography and OCT were normal, even in the older patients with OMD. The visual acuity at the initial visit varied widely, and 4 patients had 20/20 or better. These patients with good visual acuity showed preserved function in tiny foveal area, surrounded by dysfunctional macula, which was detected by subjective cone sensitivity profile. The wave shape of FERG often showed the deporalizing pattern, suggesting selective disturbance of off-bipolar cell pathway. While all patients showed a loss of cone sensitivity only in the macula, 28 patients (70%) had preserved normal rod sensitivity in the macula.

Since many of our patients with OMD were misdiagnosed as having any of several other diseases, such as psychological eye problems, amblyopia, optic nerve or central nerve problems, we have to keep in mind the existence of OMD in clinical situations.

NOTES:
DOCOSAHEXAENOIC ACID (DHA) SUPPLEMENTATION IN X-LINKED RETINITIS PIGMENTOSA (XIRP)

Rand Spencer, M. D.
Retina Foundation of Southwest and
UT Southwestern Medical Center
Dallas, Texas

Introduction:
A four-year, prospective, placebo-controlled trial was designed to determine whether docosahexaenoic acid (DHA) supplementation in patients with X-linked retinitis pigmentosa (XLRP) influences the rate of disease progression as measured by the electroretinogram (ERG).

Methods:
Male patients (mean age = 16 yr.; range 4-38 yr.) were randomized to capsules containing DHA-enriched oil (400 mg DHA/day; n=23) or a corn/soy oil placebo (n=21).

Results:
Initial RBC-DHA concentrations were 35% lower than those of age- and gender-matched normal subjects (mean + SD = 27± 5 ug/ml vs. 41 ± 6 ug DHA/ml). RBC-DHA concentrations increased an average of 2.5-fold (70 ± 20 ug DHA/ml; range = 35.9 – 101.4 ug/ml) upon supplementation and remained elevated to trial conclusion. The annual rate of decline of cone ERG amplitude to 31-Hz flicker was inversely correlated with RBC=DHA concentrations (r = -0.40; p = 0.018). There was less change in fundus appearance between Year 0 and Year 4 in the DHA group than in the placebo group (p = 0.04).

Conclusion:
These results suggest a beneficial role for long-term nutritional supplementation with DHA in retarding the functional loss associated with XLRP.

NOTES:
DIABETES

Moderators: Prof. Peter Kroll, and Bart LaFaut, MD

Genetics of Diabetic Retinopathy .............................. 8:00-8:30
Mariano Taverna, MD (Prof. Jean Louis Selam)

Pharmacotherapy of Diabetic Retinopathy ..................... 8:30-9:00
Prof. Eva Kohner

Diffuse Diabetic Macular Edema: Clinical Results .......... 9:00-9:15
of Intravitreal Injection of Triamcinolone Acetonide
Prof. Ingrid Kreissig

Multi-Center Trial of Intravitreal Steroid for Diabetic ........ 9:15-9:45
Macula Edema
Andrew Pearson, MD

Lisinopril Effects on Blood Flow & Blood Retinal- ........ 9:45-10:00
Barrier in Type I Diabetes
J. Wallace McMeel, MD

BREAK/EXHIBITS OPEN ............................................ 10:00-10:30

PAUL KAISER AWARD II

Vascular Plasticity in the Retina and Choroid ................. 10:30-11:30
Prof. Wallace Foulds

VITREO—RETINAL SURGERY III

Moderators: Glenn Wing, MD and Jerry Sebag, MD

Macular Translocation ........................................... 11:30-11:45
Carl Claes, MD

OCT & Angiographic Findings after Macular Translocation ... 11:45-12:00
Hiroko Terasaki, MD

SLO Microperimetry in Macular Surgery ....................... 12:00-12:15
Prof. Marie-Jose Tassignon

3-D OCT/SLO in Vitreo-Retinal Disease ....................... 12:15-12:30
Prof. Akitoshi Yoshida

Radial Optic Neurotomy in Ischemic CRVO ................... 12:30-12:45
Adiel Barak, MD

Prognosis of Surgery in Epi-retinal Membrane 2° .......... 12:45-13:00
to a Retinal Tear or a Rhegmatogenous RD
Prof. Catherine Gribomont

ADJOURNMENT
Diabetic eye disease and its complications, especially diabetic retinopathy (DR), are a leading cause of blindness and visual dysfunction in adults in economically developed societies. Several recent studies have provided evidence that good diabetes control is important to prevent DR. However, some groups of individuals develop DR despite good control and others escape DR despite poor control. This suggests the role of genetic factors in susceptibility to DR. Moreover, epidemiological studies, as the Diabetes Control and Complications Study (DCCT), have showed a high familial transmission for DR, especially for advanced forms. In fact, DR, a complex and heterogeneous syndrome, is a consequent of multiple interactions between environmental and genetic factors. The principal physiopathological pathways in DR are activation of protein kinase C (PKC), angiogenic factors, growth factors, polyol pathway, oxidative stress and accumulation of advanced glycosylated end-products (AGEs). Using different genetic approaches including gene candidate studies of association and functional genetic studies, we and many other authors have suggested the importance of several genes in the pathogenesis and risk of DR. The most important genes are aldose reductase (ALR2), endothelial and inducible nitric oxide synthases (eNOS and iNOS), and vascular endothelial growth factor (VEGF). The most important polymorphism associated with DR is a (A-C)n dinucleotide repeat polymorphic marker in the 5' regulatory region of the ALR2 gene. In the next future, the genetics arena of the DR will be greatly boosted by emergent revolutionary technologies as cDNA and protein microarrays, quantitative real-time PCR and laser capture microdissection (LCM). Finally, the identification of strong genetic risk factors for RD, especially for severe forms, could be employed on: 1) better prevention and earlier treatment among high risk individuals, and 2) implementation of novel pharmacological therapies (for example, specific nitric oxide synthase inhibitors).
Abstract

PHARMACOTHERAPY OF DIABETIC RETINOPATHY

Prof. Eva M. Kohner
St. Thomas’ Hospital
London, England

Diabetic retinopathy remains an important cause of visual loss, in spite of laser treatment being available now for over 30 years. More recent work indicates that both the incidence and the progression of retinopathy can be reduced by strict control of blood glucose and blood pressure. The Diabetes Control and Complications Study showed that in type 1 diabetic patients strict glucose control reduced the progression by 60%, and this good control achieved had a long term effect, even after loosening up of the control. The UK Prospective Diabetes Study (UKPDS) showed similar effect in type 2 diabetics. It showed that for every 1% reduction HbA1c there is a 25% reduction in the need for photocoagulation.

Similarly the UKPDS showed that tight blood pressure control, aiming at blood pressure below 150/90 mm. had a significant effect on reducing need for photocoagulation by 37%. The effect of beta-blockers and ACE inhibitors was similar, it was the blood pressure reduction which matters.

At present neither treatment nor addition of laser will completely prevent visual loss. However new therapies are now under trial and these may be of value. The ones to be discussed are the PKC beta 2 inhibitors, ACE Receptor inhibitors, and growth hormone and growth factor inhibitors.

It is hoped that in the not too distant future we will be able to prevent visual loss in diabetic patients.

NOTES:
DIFFUSE DIABETIC MACULAR EDEMA: CLINICAL RESULTS OF INTRAVITREAL INJECTION OF TRIAMCINOLONE ACETONIDE

Prof. Ingrid Kreissig  
Department of Ophthalmology,  
University of Mannheim  
Germany

Introduction:
Triamcinolone acetonide has been used as treatment for exudative, proliferative and inflammatory diseases. The subsequent study was undertaken to assess whether an intravitreal injection of crystalline triamcinolone acetonide may be effective in reducing diffuse diabetic macular edema and to evaluate the clinical outcome in relation to visual acuity and intraocular pressure.

Methods:
The prospective non-randomized comparative clinical interventional study included 15 patients (15 eyes) who received an intravitreal injection of 25 mg crystalline triamcinolone acetonide as treatment of diffuse diabetic macular edema. Follow-up time was 5.21 ± 4.90 months. The study group was compared with a control group of 16 patients who underwent macular grid laser coagulation.

Results:
In the study group, visual acuity increased significantly (p=0.004) from 0.08 ± 0.06 at baseline to a maximum of 0.16 ± 0.13 during the follow-up. Nine (90%) of 10 eyes with a follow-up period of more than 1 month gained in visual acuity. In the control group, visual acuity did not change significantly. In the study group, intraocular pressure increased marginally significantly (p=0.063) from 16.1 ± 3.4 mm Hg to a mean maximal value of 20.40 ± 6.6 mm Hg, and decreased marginally significantly (p=0.063) to 17.10 ± 1.5 mm Hg at study end.

Conclusions:
Intravitreal injection of 25 mg crystalline triamcinolone acetonide may be beneficial in increasing visual acuity in patients with clinically significant diffuse diabetic macular edema.

NOTES:
MULTI-CENTER TRIAL OF INTRAVITREAL STEROIDS FOR DIABETIC MACULAR EDEMA

P. Andrew Pearson, M.D.
Department of Ophthalmology,
University of Kentucky

Purpose:
A multi-center, randomized, controlled clinical trial was conducted to investigate the use of a three-year sustained release fluocinolone acetonide intravitreal implant in patients with diabetic macular edema.

Methods:
Eighty patients were randomized into one of three study treatments: 2 mg implant (n=11), 0.5 mg implant (n=41) or ‘standard of care’ (n=28) consisting of either macular grid laser or observation. All patients had persistent macular edema despite at least one macular laser procedure at least 3 months prior to randomization. Fundus photographs and fluorescein angiograms were assessed by a centralized masked reading center.

Results:
After 1 year, more patients receiving a 0.5 mg implant than SOC, had complete resolution of edema at the center of the macula. This was statistically significant (p=0.047). More patients in the 0.5 mg group had improvement in retinal thickening at the center of the macula than in the SOC group (p=0.003). At one year, more patients in the SOC lost 15 letters (14.3% vs. 4.7%) and more 0.5 mg patients gained 15 letters (19.5% vs. 7.1%); however this was not statistically significant. Complications in the implant arm included elevation of IOP and cataract.

Conclusions:
The fluocinolone implant reduces retinal thickening in diabetic macular edema. Patients will be followed for an additional 3 years.

NOTES:
EFFECT OF LISINOPRIL ON RETINAL BLOOD FLOW AND
BLOOD-RETINAL BARRIER PERMEABILITY IN TYPE I DIABETES

J. Wallace McMeel, MD
Schepens Retina Associates Foundation for Clinical Research
Boston, Massachusetts

While it is well known that ACE Inhibitor therapy is effective in the treatment of diabetic nephropathy, its effectiveness in the treatment of diabetic retinopathy has only recently come to light. It has also been clearly shown that retinal blood flow is decreased in patients with insulin-dependent diabetes when minimal or no retinopathy is present and that the decrease becomes more pronounced with time. We therefore conducted a randomized, double-masked, placebo-controlled, parallel study of the effect of the ACE Inhibitor Lisinopril on retinal blood flow and retinal blood-retinal permeability on 12 patients with Type I diabetes using the Canon CLBF 100 laser Doppler instrument and the Ocumetrics Fluorotron Master. Average age was 30 years; average duration of diabetes was 10 years; and average length of follow-up was 11.4 months. At entry, 3 patients had mild retinopathy, and 9 had no retinopathy. In the Placebo Group, retinal blood flow decreased on an average by 15.6%; in the Lisinopril Group, the flow increased by 14.6%, a significant difference (p = 0.027, Mann-Whitney U test). We also found that the change in the amount of fluorescein leakage into the vitreous was directly related to the changes in blood flow.

NOTES:
VASCULAR PLASTICITY IN THE RETINA AND CHOROID

Prof. Wallace S. Foulds
Glasgow, Scotland

It is generally held that after embryogenesis is complete the human retinal and choroidal vascular systems remain relatively unchanged throughout life requiring neither angiogenic nor angioinhibitory factors for their maintenance. It is only in diseased states such as those characterised by neovascularisation that a role for angiogenic factors is generally accepted.

An alternative scenario is that throughout life the maintenance of apparently unchanging ocular vascular systems requires a continuous but balanced input of angiogenic and angioinhibitory factors. Evidence to support this view will be presented based on clinical observation and upon experimental data supported by clinico-pathological correlates.

NOTES:
MACULAR TRANSLOCATION

Carl Claes, MD
Antwerp, Belgium

Purpose:
The aim of this study is to evaluate the functional outcome in a group of patients treated with full macular translocation (FMT) with 360-degree retinotomy for treatment of age-related macular degeneration (ARMD) with subfoveal choroidal neovascularization.

Study Design:
Consecutive interventional case series.

Methods:
Consecutive eyes (50 patients) with ARMD and subfoveal neovascularization who underwent a FMT in our department from January 1999 to July 2000 are included in this study. Compensatory muscle surgery, as described by Eckardt and associates, was performed on all the eyes. The median follow-up is 21 months (range, 12 to 36; SD, 5.4).

Results:
Best-corrected postoperative visual acuity (BCVA) was improved by 2 or more Snellen lines in 33 eyes (66 %) and remained stable (+/- 1 line) in 14 eyes (28 %). Only 3 eyes (6 %) experienced a deterioration of the BCVA of 2 or more lines. The final BCVA was 20/50 or better in 32 % of the cases; only 8 eyes (16 %) had a final BCVA < 20/200. 34 (68 %) patients are able to read newspaper print (3.3/10) with normal (+ 3 diopters to + 4 diopters) or increased (+ 5 diopters to + 8 diopters) reading ads. Other patients are able to read with magnifying systems. Complications included proliferative vitreoretinopathy (PVR) in 9 eyes (18 %) recurrent choroidal neovascularization in 5 eyes (10 %), diplopia in 3 eyes (6 %), choroidal hemorrhage in 2 eyes (4 %), macular hole in 1 eye, and temporary hypotony in eye.

Conclusions:
As 68 % of the patients in the study group regained reading vision with reading glasses, FMT can be considered an effective approach in cases of subfoveal choroidal neovascularization. Further investigations are necessary to determine which patients will have the most benefit from this complex therapeutic method.
OPTICAL COHERENCE TOMOGRAPHY AND FLUORESCEIN ANGIOGRAPHY AFTER MACULAR TRANSLOCATION

Hiroko Terasaki, MD, Prof. Yozo Miyake
Nagoya University School of Medicine
Department of Ophthalmology

Purpose:
To determine the configuration of the macula by optical coherence tomography (OCT) and the vascular integrity of the macula by fluorescein angiography (FA) after macular translocation (MT) surgery for subfoveal choroidal neovascularization (CNV).

Methods:
OCT and FA were performed 10 months after MT surgery with a 360° degree retinotomy in 23 consecutive eyes. The diameter of the CNVs ranged from 0.3 to 2.6 disc diameters, and the angle of rotation of the retina ranged from 11° to 45°.

Results:
The pre-operative visual acuity (BCVA) ranged from hand motion to 20/100, and the post-operative BCVA ranged from 20/667 to 20/25. OCT images demonstrated a concave foveal configuration after surgery in all 23 eyes with the mean foveal thickness of 150 μm. FA showed various degree of fluorescein leakage with a pattern similar to cystoid macular edema in 15 of 23 eyes (70%).

Conclusion:
The newly located macula had normal macular configuration with normal thickness, but there was a disparity between the OCT and FA findings. A cystoid macular edema-type of leakage appeared to be compensated for by the relatively well-functioning RPE where the macula was newly located.

NOTES:
Abstract

PREDICTIVE VALUE OF SLO MICROPERIMETRY IN MACULAR SURGERY

Prof. Marie-Jose Tassignon, Erica Smets, MD
University Hospital Antwerp
Antwerp, Belgium

Aim:
Macular surgery is commonly performed for many vitreo-retinal disorders. However, there are still no tests to predict visual outcome or to measure the quality of vision after surgery. SLO microperimetry can be useful as a predictive tool for visual recovery after surgery and as a method to objectively evaluate post-operative quality of vision.

Methods:
Patients who underwent macular surgery for macular pucker, macular hole or submacular neovascular membranes were examined pre- and post-operatively with the SLO microperimetry program.

Results:
Pending

Conclusion:
The SLO is very useful tool to predict visual recovery after vitreo-macular surgery and as a method to objectively evaluate quality of vision post-operatively.

It should be mentioned, however, that the measurement of the Stiles Crawford orientation of photoreceptors would be the next step to focus on in order to better evaluate vision after macular surgery.

NOTES:
Abstract

RON IN ISCHEMIC CRVO—PRELIMINARY RESULTS

Adiel Barak, MD
The Tel Aviv Medical Center
Israel

Purpose:
Central retinal vein occlusion (CRVO) is a leading cause of permanent visual blindness. Recently, surgical decompression of the scleral outlet, termed radial optic neurotomy (RON), has been advocated as an effective treatment for ischemic CRVO. The authors performed RON in patients with ischemic CRVO to try and improve visual results in these patients.

Methods:
RON was performed on 8 consecutive patients with ischemic CRVO. The surgical technique included pars plana vitrectomy, posterior cortical vitreous separation and perforation of the optic nerve tissue using an MVR blade.

Results:
Radial optic neurotomy was performed successfully in all 8 patients. All patients had clinical improvement as determined by fundus examination, photography, and fluorescein angiography. Postoperative visual acuities improved in 4 (50%) patients. Complications include retinal detachment which developed in one patient.

Conclusions:
Surgical decompression of CRVO via RON is a technically feasible and initially safe procedure that is associated with improved visual acuity in ischemic CRVO.

NOTES:
Purpose:
We evaluated Age-Related Maculopathy (ARM), idiopathic Macular Hole (MH), and Macular Edema (ME) findings using a new imaging device that simultaneously combines OCT and SLO images in a single instrument.

Methods:
Fifty eyes (30 patients) with ARM, 30 eyes (28 patients) with MH, and 30 eyes (20 patients) with ME were studied. The new system can operate in a transversal and yields Humphrey’s OCT images. The C-scan OCT images are thin cross-sections of retinal and subretinal structures at a chosen depth.

Results:
This new device precisely demonstrated the structures of the retinal layers and the pathologies in the retina, especially in patients with ARM, MH, and ME. In some cases, clear separation between the outer layers of rod and cone layer and retinal pigment epithelium layer was detected.

Conclusions:
This new technology for the first time enabled us to observe vitreo-retinal structures in 3-D fashion. This technique is useful to analyze the retina and other ocular structures in a manner similar to how computed tomography and magnetic resonance imaging are used for other organs in the human body.

Other Authors:
Satoshi Ishiko and Yoshitaka Horikawa

NOTES:
PROGNOSIS OF SURGERY IN EPIMACULAR MEMBRANES SECONDARY TO A RETINAL TEAR OR A RHEGMATOGENOUS RETINAL DETACHMENT

Anne-Catherine Gribomont, MD  
Cliniques Universitaires Saint-Luc,  
Ophthalmology Department  
Brussels, Belgium

Introduction:  
The prognosis of epimacular membranes secondary to a retinal tear or a rhegmatogenous retinal detachment -treated or not- seems less favorable than that of idiopathic macular pucker's. The aim of the study is to analyze the functional results and retinal complications of the surgery in such secondary epimacular membranes, in comparison with idiopathic membranes.

Methods:  
This retrospective study compares a consecutive series of 28 cases of secondary membranes and 63 cases of idiopathic membranes with at least a 3-month follow-up. The main outcomes are the visual acuity changes 3 to 6 months after surgery - taking into account the lens status - and the incidence and characteristics of primary and recurrent retinal detachments occurring after surgery.

Results:  
For the secondary membranes, a two-line or more visual acuity improvement is obtained in 36% (4/11) of eyes with progressive nuclear sclerosis, 50% (3/6) of primary pseudophakic eyes, and 80% (8/10) of eyes with a clear lens. With a mean follow-up of 8.7 months, the incidence of retinal detachment is 25% (7/28). Among the 7 retinal detachments, only one is typically iatrogenic.

For the idiopathic membranes, a two-line or more visual acuity improvement is obtained in 50% (25/51) of eyes with progressive nuclear sclerosis, and 100% (12/12) of eyes with a clear or operated on lens. With a mean follow-up of 9.2 months, the incidence of retinal detachment is 1.5% (1/63).

Conclusion:  
Our results confirm that the prognosis of macular pucker's secondary to a retinal tear or detachment is mediocre compared to that of idiopathic membranes, with a very high incidence of primary or recurrent retinal detachments after surgery.
POSTERS

Optic Disc Abnormality Disclosed by OCT
Sachiko Hamada, MD

Recurrent CME Induced by Topical Latanoprost in Pseudophakic Eyes
Mikki Arai, MD

Pneumatic Retinopexy and Patients’ Aerial Transport
Carlos Heredia Garcia, MD

Diabetic Vitreopathy
Stephan Dunker, MD

Retinal Oxygen Saturation in BRVO
Mateusz Scibor, MD

Effects of ICG and Trypan Blue on Human RPE
John Gonder, MD

A Comparison of PDT and TTT
David Maberley, MD

Schisis-Like Retinal Detachment After Blunt Ocular Trauma in a Patient with Congenital Optic Pit
Carsten Meyer, MD, Eduardo Rodrigues, MD, Stefan Mennell, MD

Unsealed Sclerotomy After Intravitreal Injection with a 30-Gauge Needle
Eduardo Rodrigues, MD, Karsten Meyer, MD, Stefan Mennell, MD, Joerg Schmidt, MD

Clinical Follow-Up After Intravitreal Triamcinolone Acetonide
Stefan Mennell, MD, Karsten Meyer, MD, Eduardo Rodrigues, MD, Joerg Schmidt, MD
OPTIC DISC ABNORMALITY DISCLOSED BY OPTICAL COHERENCE TOMOGRAPHY (OCT)

Sachiko Hamada, M. D.
Hamada Eye Clinic
Nara City, Japan

In 1995 a 67-year-old woman presented with abnormal feeling in the left eye. Retinal edema was demonstrated in the macular vascular arcades without evidence of optic disc pits on ophthalmoscopy and fluorescein angiography. Visual acuity was 1.2 with normal intraocular tension in each eye.

In 1999 the nasal retina became edematous. OCT disclosed retinoschisis in the edematous macula and nasal retina, retinal detachment in the fovea, and further abnormality of the optic disc: abnormal small glial tissue, cystic cavities beneath the inner-most membrane which seemed to be connected with the area of retinoschisis.

It is speculated that liquid vitreous might go through the membrane inside the cystic cavities and then into the retina, which developed retinoschisis.

NOTES:
RECURRENT CYSTOID MACULAR EDEMA INDUCED BY TOPICAL LATANOPROST IN A PSEUDOPHAKIC EYE

Mikki Aria, M. D. and Ryoji Yamakawa, M. D.
Department of Ophthalmology,
Kurume University School of Medicine
Furume City, Fukuoka, Japan

Purpose:
To report reversible cystoid macular edema (CME) in a pseudophakic eye associated with topical latanoprost therapy.

Methods:
A 54 year-old man with ocular hypertension underwent cataract surgery with anterior vitrectomy and IOL fixation in the ciliary sulcus in the left eye 18 months previously because of spontaneous subluxation of the lens. He was followed with ophthalmic examinations including optical coherence tomography (OCT).

Results:
CME with decreased vision to 20/60 developed in the left eye 8 weeks after starting topical latanoprost. Five weeks after discontinuing latanoprost, the macula flattened and vision recovered. To control intraocular pressure, latanoprost was prescribed again. Eight weeks after restarting it, CME developed with decreased vision to 20/25. It resolved again upon discontinuing latanoprost within 7 weeks. The visual acuity recovered completely. No change was detected in the right eye, regardless of whether latanoprost was used or not.

Conclusion:
Latanoprost was related to CME formation and resolution in this pseudophakic eye.

NOTES:
Abstract

PNEUMATIC RETINOPEXY AND PATIENTS AERIAL TRANSPORT

Carlos Dante Heredia Garcia, M. D., PhD.
Centro de Oftalmología Bonafonte
Barcelona, Spain

Introduction:
Air travel in both pressurized airliners and non-pressurized airplanes is safe for patients with pneumatic retinopexy. Nevertheless, consideration should be given to difference between altitudes of the place where retinopexy is performed and the final destination of the patient after the procedure.

Methods:
There are many physical laws that influence the behavior of expansive gases in the eye. We all know that these substances are insoluble in water, and can therefore remain in the vitreous cavity for a longer time than in air. We also know that when a bubble of SF6 is injected, its volume becomes 1.5 times greater in 24 hours and reabsorbs in 14 days. C3F8 on the other hand, increases in volume four times in 72 hours, and total re-absorption takes place in about 25 days. All this is under normal conditions. However, based on the Boyle-Mariotte law there are modifications in the volume of gas bubbles in connection with changes in altitude. A decrease in barometric pressure produces an increase in the volume of gas in the eye, which eventually leads to high intraocular pressure (IOP) and possible adverse consequences.

Results:
A patient who is going to travel to one place at an altitude higher than 2,000 m. with expansive gases will have 40% volumetric increase of the tamponade eye and if the procedure is performed at a higher altitude and the patient then travels to a place near sea level, the inverse effect will occur. The volume of the gas diminishes by 40% resulting in very poor efficacy of the tamponade. Traveling by air carries no risk for patient with pneumatic retinopexy by the presence of compressed air inside the cabins of the airplane.

Conclusion:
Instead take into account the altitude of the place where the patient with pneumatic retinopexy is going to stay. Transport by private planes and small private planes non-pressurized but flying below 3,000m can be considered supremely safe.
DIABETIC VITREOPATHY

Stephan Dunker, MD
Troisdorf, Germany
Prof. Jürgen Faulborn, Graz, Austria

Introduction:
Diabetic vitreopathy is one of the major contributors to diabetic retinopathy. In this study, the role of vitreous in diabetic eyes is investigated histologically.

Methods:
26 eyes were obtained from 19 patients with type II diabetes. The duration of diabetes in these patients ranged from 8 to 26 years (average 17 years). The eyes were embedded in celloidin and examined histologically using light microscopy, transmission electron microscopy and scanning electron microscopy.

Results:
Vitreous in diabetic eyes exhibits delayed senile degeneration in case of early onset of diabetes. The vitreous shows intensified staining in the vitreous cortex. Posterior vitreous detachment is often incomplete without collapse. The fibers are coarse and aggregated in the centrally located cortical region of the vitreous. We could find collagen fibers that showed traction to the fovea with associated thickening of the retina. All these structural changes were not demonstrated in non-diabetic eyes.

Conclusion:
The vitreous in diabetic patients is morphologically different from normal vitreous. One possible explanation of the vitreous changes is non-enzymatic glycation of the vitreous resulting in the findings described above.

NOTES:
Abstract

RETINAL OXYGEN SATURATION IN BRVO

M. Scibor 1, H. Wenkel 1, G. Michelson 1, D. Schweitzer 2, M. Hammer 2, J. Harazny 1

Purpose:
To non-invasively measure oxygen saturation in retinal arteries and retinal veins and systemic oxygen saturation in capillaries of normals and patients with BRVO.

Methods:
The determination of retinal oxygen saturation was performed using the Imaging Spectrometer. The basis for the non-invasive estimation of the oxygen saturation is different extinction spectra of hemoglobin and oxyhemoglobin. The measurements of the reflectance spectra were performed in a wavelength range from 400 nm to 700 nm. The retinal oxygen saturation was measured with an entrance slit of 1.5 mm, spectral resolution of < 2m, and local resolution s < 7.8m.

The determination of capillary systemic oxygen saturation was performed with blood gas analyser Radiometer ABL 510.

We measured 72 healthy eyes, 18 eyes with retinal vein occlusion of non-ischemic type, and 11 eyes with retinal vein occlusion of ischemic type. The type of retinal vein occlusion (ischemic or non-ischemic) was determined by fluorescein angiography.

Results:
In normals, the retinal oxygen saturation was on average 91 + 5% in arteries and 57 + 8% in the veins. In patients with non-ischemic retinal vein occlusions the oxygen saturation was on average 88.7 + 3.1% in retinal arteries and 57.3 + 7.6% in retinal veins. In patients with ischemic retinal vein occlusions the oxygen saturation was on average 84.7 + 5.2% in retinal arteries and 36.1 + 6.9% in retinal veins.

The comparison with the oxygen saturation in normal eyes shows significant decrease of oxygen saturation in arteries as well as in veins in eyes with ischemic retinal vein occlusion of (p<0.001). The capillary systemic oxygen saturation in patients with venous retinal occlusion was in average 95 + 3%.
Conclusions:
In eyes with ischemic BRVO we found a significant decrease of local oxygen saturation in retinal veins and retinal arteries (p<0.001). In eyes with non-ischemic retinal vein occlusion we didn’t find a significant difference of local oxygen saturation in the retinal arteries and retinal veins in comparison to normals (p=0.09 in arteries; p=0.21 in veins).
In the systemic capillary oxygen saturation we found no significant difference between normals and patients with retinal vein occlusions (p=0.51).

1: Dept. of Ophthalmology, University Erlangen–Nürnberg, Erlangen
2: Dept. of Ophthalmology, University Jena, Germany

NOTES:
Abstract

A COMPARISON OF THE INDOCYANINE GREEN AND TRYPAN BLUE ON CULTURED HUMAN RETINAL PIGMENT EPITHELIAL CELLS

Gonder, MD, Hutnik, MD, Gale, MD, and Mao MD, Ivey Eye Institute
London, Ontario, Canada

Introduction:
In macular hole surgery, ICG dye is used to assist in internal limiting lamina removal. Recent clinical studies have suggested the use of ICG adversely affects visual outcome. Indeed, cell culture studies have demonstrated that high concentrations of ICG dye are cytotoxic. However, clinically useful concentrations of ICG dye have not been assessed with regards to their cytotoxicity in cell culture. Furthermore, trypan blue dye has recently been used for anterior segment surgery and is being reviewed for retinal surgery.

Methods:
We carried out in vitro studies using human RPE cell cultures to assess the clinical utility of appropriate concentrations of both ICG and trypan blue dyes.

Results:
ICG dye demonstrated a dose-dependent and exposure-dependent toxicity. However, a 60 second exposure with ICG dye at a concentration of 0.5 mg/ml, was not toxic in our cell cultures.

Trypan blue dye was found not to be toxic to the RPE cultures at any concentration over any period of time exposure.

Conclusion:
ICG dye demonstrated more toxicity than trypan Blue in human RPE cell cultures. However, a concentration of ICG dye (0.5 mg/ml for 60 seconds) was not toxic to human RPE cells in vitro and, as a result, could be considered as a useful surgical tool.

NOTES:
A COMPARISON OF PDT AND TTT

David A. L. Maberley MD, FRCSC, MSc (Epid)
Department of Ophthalmology, University of British Columbia, Vancouver, B.C.

Patrick Ma MD, FRCSC¹, Angela Chang BSc¹

Hall Chew MD
University of Toronto, Department of Ophthalmology, Toronto, Ontario

Hussein Hollands MSc (Epid), BSc
Alan Maberley MD, FRCSC¹

Background:
The purpose of this study was to compare photodynamic therapy (PDT) with sub-threshold diode laser (transpupillary thermo-therapy/TTT) for the treatment of sub-foveal choroidal neovascularization secondary to age-related macular degeneration.

Methods:
Subjects with sub-foveal choroidal neovascularization secondary to age related macular degeneration (AMD) were offered photodynamic therapy (PDT) treatment as an initial intervention. If subjects declined PDT, then TTT treatment was offered.

Results:
115 consecutive subjects were evaluated. The primary outcome measure was visual acuity, but subjects were also compared on the basis of lesion size and angiographic leakage. Baseline comparisons between groups showed significant differences for pre-treatment visual acuity, lesion size, and lesion composition. Univariate analysis of outcomes demonstrated equivalency between treatment groups for lesion size, angiographic activity and visual acuity. On multivariate analysis, the two treatment groups were equivalent on the basis of final visual acuity when controlling for age, lesion categorization, and pre-treatment visual acuity. Predominantly classic lesions were associated with poorer visual outcomes.

Conclusions:
The transpupillary thermo-therapy and PDT treatment groups did not have significantly different final visual acuities. Patients with predominantly classic lesions tended to have poorer visual outcomes.

¹: Department of Ophthalmology, University of British Columbia, Vancouver, B.C.
2: University of Toronto, Department of Ophthalmology, Toronto, Ontario
SCHISIS-LIKE RETINAL DETACHMENT AFTER BLUNT OCULAR TRAUMA IN A PATIENT WITH CONGENITAL OPTIC PIT

Carsten H. Meyer, MD, Eduardo B. Rodrigues, MD, Stefan Mennel, MD
Department of Ophthalmology
Phillips-Universitat
Marburg, Germany

Abstract

Introduction:
Patients with congenital optic pit may become symptomatic through several mechanisms, leading to serous retinal detachment. We report a case where ocular trauma may have triggered retinal detachment associated with optic pit.

Case Report:
We present a 16-year-old Caucasian girl with a 3 month history of blunt trauma to the left eye. Fundus examination revealed a retinal detachment extending from the optic disc to the whole macular region. Visual acuity was decreased to 20/30 OS, and remained unchanged 20/20 OD. Optical coherence tomography (OCT) disclosed a schisis-like macular detachment. While OCT examination demonstrated a clear separation between the inner and outer schisis-like layers, only the inner layer separation was continuous with the optic pit. B-scan ultrasound examination demonstrated communication between the vitreous Cloquet’s canal and the surface of the optic pit.

Comment:
The early onset of the retinal detachment, the adherent posterior vitreous, and the history of blunt trauma before the beginning of the detachment make reasonable the hypothesis of trauma as causative factor for retinal detachment. In conclusion, patients with congenital optic pit and posterior vitreous attachment may develop retinal detachment after blunt ocular trauma.

NOTES:
UNSEALED SCLEROTOMY AFTER INTRAVITREAL INJECTION WITH A 30-GAUGE NEEDLE

Eduardo B. Rodrigues, MD
Carsten H. Meyer, MD; Mennel S, MD; Joerg C. Schmidt, MD
Department of Ophthalmology
Phillips-Universitat
Marburg, Germany

Introduction:
Scleral incisions using a 27- or 30-gauge needle for injection of air, gases, or drugs are frequently performed in the treatment of several macular diseases. However, little is known about complications of unsutured 27- or 30-gauge incisions. We report two cases where an unsutured 30-gauge scleral incisions resulted in vitreous incarceration in an unvitrectomized eye, and hypotony with choroidal detachment in a vitrectomized eye.

Report of cases:
In the first case, a 55-year-old patient was diagnosed with massive submacular hemorrhage secondary to age-related macular degeneration. He underwent tissue plasminogen activator (TPA) intravitreal injection with a 30-gauge transconjunctival needle. On the first post-operative day, injection of 0.3ml SF6 using also a 30-gauge needle was performed. Five weeks later pars plana vitrectomy (PPV) for intravitreal blood removal was indicated. The unsutured 30-gauge incision was open, without any signs of wound healing process, and remarkable vitreous incarceration was visualized.

In the second case, a 29-year-old patient with panuveitis underwent PPV for vitreous opacities. Because of a post-operative diffuse macular edema, intravitreal injection of triamcinolone with a 30-gauge needle was performed. On the third post-operative day the intraocular pressure decreased to 0 mmHg, and choroidal detachment was visible in the infero-temporal quadrant. The previous 30-gauge incision site at the 12 o’clock position was still open without signs of wound healing process.

Comments:
We report complications in two cases where a 30-gauge needle was used for intravitreal injections. Efforts to decrease the size of the pars plana incisions and to avoid scleral suturing are arising. Self-sealing sclerotomies have already been reported to be safe and efficient. Recently, an unsutured 25-gauge system for PPV has been introduced. Our report of cases draws attention to the occurrence of wound-related complications after the small, unsutured 30-gauge scleral incisions.
CLINICAL FOLLOW-UP AFTER INTRAVITREAL TRIAMCINOLONE ACETONIDE

Stefan Mennel, MD
Carsten H. Meyer, MD; Eduardo B. Rodrigues, MD;
Joerg C. Schmidt, MD
Department of Ophthalmology
Phillips-Universitat
Marburg, Germany

Introduction:
Intravitreal Triamcinolone acetonide (TA) is a new therapy to treat several macular diseases including macular edema and age-related macular degeneration. There are several examination methods described in the literature to follow the clinical effects of intravitreal TA. Visual acuity (VA), fundus photography, fluorescein angiography (FA), linear optical coherence tomography (OCT) and three-dimensional (3D) OCT are compared in this study and the advantage of each examination is pointed out.

Patients and Methods:
TA was injected intravitreal in 16 patients with macular edema secondary to central retinal vein occlusion (CRVO), branch retinal vein occlusion (BRVO), uveitis and diabetic maculopathy. Routine VA and FA examination were performed in all cases. Additionally, fundus photography, linear OCT and 3D OCT were conducted pre- and post-operatively.

Results:
VA improved in 12 patients. Pre-operative FA examination demonstrated extensive macula leakage in all cases. While in eight cases a marked leakage reduction was observed post-operatively on FA, in four cases the quantification of leakage was not possible. Fundus photography allowed appropriate visualization of the macular changes in only four cases. Linear OCT showed a reduction of retinal thickness from 380-620µm to 168 – 450µm post-operatively. In two cases no significant differences could be documented. Because 3D OCT represents a large area of 6000µm in diameter, macular edema changes could be clearly observed in all cases.

Conclusion:
Although in some cases VA did not improve, other examinations such as linear and 3D OCT demonstrated a high efficacy to detect macular changes after TA injection. 3D OCT is the most sensitive and precise examination to demonstrate the therapeutic effects of intravitreal TA application.
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SIGNATURE: ______________________________________ DATE: ____________________________
### Sunday, July 6, 2003

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<td>Carl Claes, M.D.</td>
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<td>1615</td>
<td>Innovations in Vitreo-Retinal Surgery</td>
<td>Eugene de Juan, M.D.</td>
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<td>Effects of ICG and Trypan Blue on Human RPE</td>
<td>John Gonder, M.D.</td>
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<td>Fluorescence of Optic Nerve and Macula after ICG-Assisted Macular Surgery</td>
<td>Jean Francois, M.D.</td>
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### Monday, July 7, 2003

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<td>Pathogenesis of Age-Related Macular Degeneration</td>
<td>Gregory Hageman, PhD</td>
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<td>Membranous Debris and Basal Laminar Deposits Determine the Clinical Presentation of Early Age-related Macular Degeneration</td>
<td>Shirley &amp; John Sarks, PhD</td>
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<td>ICG Angiography Classification in ARMD</td>
<td>Prof. Gabriel Coscas</td>
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<td>0920</td>
<td>Does a Macula-Off Retinal Detachment Influence the Pathogenesis of AMD? A Long Term Follow-up</td>
<td>Prof. Peter Kroll</td>
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<td>Transpupillary Thermotherapy of Occult Choroidal Neovascularization</td>
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