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CELEBRATING OUR 20TH ANNIVERSARY (20 YEARS) OF PUBLICATION

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GAROUFALIS, GEORGIJEVSKI and KOKLANIS: Long-Term Vision Outcomes of Conventional Treatment of Strabismic and Anisometric Functional Amblyopia

- MEETING REPORT -

FARZAVANDI: EXTRAVAGANZA IN LAS VEGAS, 2006.
Strabology Report of the 2nd Pediatric Ophthalmology and Strabismus Day Directed by Kenneth Wright, MD

EDITORIAL -

ROMANO: Here We Go, Charging Full Throttle and Headlong Into the Internet. Binocular Vision and Strabismus Quarterly Stops the Presses! All But Totally, and Re-Establishes Its Old Website at BINOCULARVISION.NET for Continuing e-Publication of this Now e-Periodical

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Meeting Reported by Sonal Farzavandi, FRCS
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**A History of this scientific periodical, Binocular Vision & Strabismus Quarterly. A Celebration of Our 20th Anniversary**

The year was 1984, Parks's sub-specialty of pediatric ophthalmology, which had absorbed the previously free standing, but much less marketable subspecialty of ocular motility/strabology, in the United States, for sure, was growing rapidly. This was the era before managed care intruded. It was the very last of the good years to be a physician and/or an academician.

Our sole subspecialty periodical was the *Journal of Pediatric Ophthalmology and Strabismus* which had been founded as the *Journal of Pediatric Ophthalmology* (only) by Editor Samuel V. Abraham in 1964 (the very first year your Editor started his ophthalmology training!). With the advent of the American Association for Pediatric Ophthalmology (and [later] strabismus) in the early 70's it was now fully owned by its New Jersey publisher, Slack. With the requirement of submission of all papers from the annual AAPOS meetings, the waiting time in 1984 for publication of an article in the *JPOS* was two and a half years! Even though the journal was subsidized by the AAPOS, Slack would not enlarge the journal enough or even at all to handle this demand, in spite of significant profits from the journal. No solution to this dilemma was apparent from Slack, or elsewhere.

At that time your founding editor (FE) was running the Pediatric Ophthalmology and Strabismus Service at the University of Florida in Gainesville. Another member of the faculty there, Frank Pollack, who ran the Cornea Service, had recently started a new journal entitled "Cornea" with the help of the American, New York, arm of the French publisher, Masson.

At a departmental party at his home, he proudly showed us his new computer and office for running his journal. Realizing that our subspecialty could certainly use some help with regard to publishing that backlog of 2 1/2 years for the *JPOS*, we mentioned our problem to Pollack. He said he would talk to Pierre LaHaye, at Masson in New York, who was in charge of the eye journals.

Pierre said they were very interested in another eye subspecialty journal. We agreed on undertaking the job of assembling an Editorial Board and soliciting articles as the first Editor. It didn't take long to put together an outstanding, large, inter-national Editorial Board. Alberto Ciancia and Joseph Lang were especially helpful. Everyone agreed it was a good idea. Only my old mentors refused an invitation to join the Ed Board! (I guess taking a job working for a former student is not a high priority!) They still thought it was a good idea. Excellent scientific articles were quickly volunteered by many Ed Board members. We started putting the first issue together with the help of Alvin Fayman who was to be our production manager at Masson.

Only one person in our professional community objected to *BVQ*, because he thought we were already publishing enough articles about strabismus and he didn't want to read any more. He wrote to our entire professional community in an effort to stop our efforts, but no one seconded his singular sentiments.

We titled the journal "Binocular Vision" because *BV* is what the study and treatment of strabismus is really all about. "BV" is also the first term of the title of our mentor von Noorden's esteemed textbook "Bible", so it had to be OK.

"BV", we intended, would compliment and fit in with the "JPOS", both literally and figuratively. It would not sound like a direct competitive threat - which too many of our associates were all too ready to assume anyway!

To be sure, we further staked out our strabology area by adding as a subtitle "eye movements, strabismus, and amblyopia".

We called it a "quarterly", because that was our intended publication schedule for starting, and because we think the name of a periodical, when it takes a common term for a title, needs to have another word in the title so the periodical is not confused with, and does not have to be additionally separated from, the clinical item; (i.e., when you refer to periodicals like *Ophthalmology* or *Retina* or *Cornea*, don't you often find yourself adding, "the journal" so your listener knows that you are referring to a periodical and not to a piece of anatomy or a science? But "journal" is only French for "daily", so we called it "Quarterly" which it truly is).

In early 1985 as the first issue was about to go to press, in April, Masson suddenly decided to close its American branch and sell all of its scientific periodicals, "lock, stock and barrel", to Raven Press in New York. (We suddenly felt like a professional athlete getting "traded" without choice or input.) The President/owner of Raven Press, Dr. Alan Edelson, PhD, invited us to journey to their New York offices to discuss the future of *BVQ* with him.

On arrival, Edelson first told us that Tom France, then President of the AAPOS, had just visited him *only the day before* seeking a publisher to replace Slack, who would not permit the needed expansion of the *JPOS*.

Edelson suggested that France and the AAPOS and we could and should combine the two publications into one Raven publication.

Tom and I presented this idea to both of our Editorial Boards. But our board members were most enthusiastic about having a separate journal devoted specifically to strabismus and binocular vision and only by remaining separate could we do so. Nor did sharing their journal with us go over
very well with Tom France, the JPOS Editorial staff, or the AAPOS.

That left Dr. Edelson and Raven with just us, BVQ. Edelson said that BVQ did not justify his efforts financially. Since BVQ had not even printed its first issue yet, Edelson felt no obligation to me, BVQ, or its Editorial Board members. Therefore, he said, that he would not publish BVQ but rather would simply abandon/cancel BVQ and just let us die, evaporate or whatever.

Neither I nor our Editorial Board liked that at all. After further discussions Dr. Edelson agreed to "give" the ownership and rights to BVQ to your FE. He said we could try to publish it ourselves, on our own. We had to contractually agree to do it all by ourselves and not seek or use the assistance of, or sell BVQ to, any (other) publishing house for at least five years.

So we became owner and publisher as well as the Editor.

The first major hurdle was to get the OK of our boss, the University of Florida Ophthalmology Professor and Chairman who was himself, with his wife, a medical publisher (Triad Publishing, Gainesville). Fortunately, our non compete contract with Raven would not allow him to require that BVQ be published by his company, Triad.

We found a local printer in town, Ewing Press, who printed the football programs for the University of Florida football program, (Go Gator/s) and with the help of a free lance local typesetter we set about publishing the journal. Your FE did the old cut and paste wax layout routine,[Can you remember that?!] The first issue was actually completed, printed and mailed out near the end of 1985. Volume 1 was initially called "1985" because we still had high hopes of somehow making that year our first full year of publication. However, that was not to be and the first full year of the journal was actually 1986, denoted Vol. 1, "1985-1986". Within that first year we were also to have the first of many recurring changes with printers. Ewing Press was bought out by another local printer, Marsh, and we had to break in another set of layout, typesetters and press operators.

Volume 2 was then calendar year 1987. In 1988 (Volume 3), half way through it our printer, Mr. Marsh, passed away and the firm closed down. We then went to our third local Gainesville printer in three years, Stor.er.

In early 1989, after leaving UF, and thanks to computers we took over in house production of BVQ. Fortunately, "desktop publishing" on computers had just reached the point where one did not have to be a computer engineer-whiz to do it.

So we plunged in full time, purchasing a 286-12! desktop (for about $1800!) and an HP Laser Printer (for another $1400!), which, believe it or not, has just been retired after 14 years of service although it has required repair from time to time. Unbelievable? that printer also had about 100,000 road miles on it as we trucked it back and forth between Florida and Colorado every 3 months for five years (until we moved here in 1995). We certainly have seen a number of computers (?!12+) come and go, and almost as many copy and fax machines as well during this same 14 year period. But we still use our original word processing software WordPerfect 5.0 because that is all we needed then and now.

Your FE, because of his ancient artistic bent, (alternate careers at one point were architecture and industrial or automotive design) continued as the layout man and became also the typesetter while his "better half" became chief typist as well as both the managing editor and the business manager, which included doing just about everything else except the printing. She is in fact really "the publisher."

We learned a lot and fast. In those days, it took two months of our time, truly full time, both of us, to turn out each issue. That gave us a few weeks to breath and catch up on other things in between issues. (We have gotten a lot quicker at it, but it still takes the better part of a month.)

At the end of 1989, Storler decided they suddenly needed a lot more of our money just to print the journal since we were no longer paying them for layout and typesetting. So we searched for another printer which we finally found down in Kissimmee, (near Orlando), Cody Publications, who was at that time all periodicals. They were great, printing 50 or 60 commercial publications.

Also that Spring, on recommendation, we traveled to Washington DC to personally talk to the people at the National Library of Medicine about getting into Index Medicus. It was already longer that we thought it should have been but we were soon to find our expectations not rapidly fulfilled. Nor did our visit to NIH seem to help at all, in spite of our attempt to play Washington politics.

Maybe, we thought, a more impressive title would help, so we became Binocular Vision & Eye Muscle Surgery thinking that "surgery" in the title might be a key to entry to the NLM as we could claim to be the only journal devoted to strabismus SURGERY.

Just a year later, in 1991, Mr. Cody retired and closed his printing business. One of his salesmen, a Mr. Willis, opened his own company and tried to service Cody's customers. However, as good as he was as a salesman, he was not a good printer's agent and after a couple of difficult issues, we again sought printing elsewhere.

This time we found it in the F.M.A., the Florida Medical Association. We turned to their printer in Jacksonville, Centurion Press. They did a nice job on the monthly Florida Medical Journal and they did a nice job for us. But, once again, after just a few good years the Florida Medical Society, which had
Our Objectives: Reprinted annually from the first proofs of Binocular Vision Quarterly

From The Editor

A Statement of Purpose

This journal has been conceived and is presented to fulfill current literary reporting needs we perceive. The term "we" includes, in this case, Masson Publishing USA*, an esteemed international publisher of numerous medical books and subspecialty medical journals.

First, it is inevitable that life becomes more specialized as the knowledge explosion in which we live continues. Just as general medical journals have been supplemented by specialty journals, so now must those specialty journals be supplemented by subspecialty publications. Until now, there has been no international subspecialty journal devoted specifically and exclusively to the area of binocular vision. It is this "niche" which we plan to fill. To date we have been strongly encouraged in this endeavor by many colleagues. These include the members of our editorial board, whom we specifically thank for joining us and contributing to this venture.

Second, we will cover binocular vision both horizontally and vertically - that is to say, both around the globe and from basic science researcher, orthoptist and optometrist to clinical strabologist, the better to promote communications and the progress of knowledge and patient care in this area.

Third, although North American strabologists have an easily accessible journal with no publication fees in which to publish their works, this is not true for strabologists of the other five and a half continents. We would like to provide a similarly accessible, no-fee forum for our international colleagues.

Fourth, at this moment in time, academic productivity in this area has reached a very high level.

We welcome the opportunity to serve our colleagues. At the same time we encourage them to submit their work also to the many superb national orthoptic journals and newsletters. Friends and associates have worked hard to make these publications excellent. We would like to supplement their efforts, and plan through cooperative efforts to encourage and facilitate their continued growth as well. Please join us in our endeavor.

Paul Romano, M.D.

*Masson discontinued its American operations just before the first issue of BVQ went to press. It has been published ever since by Binoculus Publishing.

created Centurion Press to print their journal, closed it, and turned the printing of its journal over to a major foreign source. [early out-sourcing!] Some employees at Centurion, who had been most helpful to us, found themselves new printing jobs and us too a new, and our current, printer, Economy Printing, Jacksonville. We have been with them ever since even though we are now retired to Colorado. Fed Ex and UPS and faxes make it easy.

In 1995, following the introduction of a European journal entitled simply Strabismus two years earlier, we felt our colleagues could hardly accuse us of threatening them anymore and replaced the "Surgery" in our title to become what we still are today, Binocular Vision and Strabismus Quarterly. We also finally officially retired from Florida and clinical practice, to the Rocky Mountains.

The last major chapter in our history to date, was our admission, finally, after 14 years, to Medline and Index Medicus in the middle of 1998. This was followed almost immediately by admission to Excerpta Medica and EM Base. This was at least largely the result of the good offices of BV&SQ Editorial Board members Larry Tychsen and David Guyton.

[For the most complete index, however, of what has appeared in BVQ over the past 17 years, including the dozen before we made the NLM grade we still compose and publish our own Index Binoculus. We shall continue to do so because the NLM is only interested in indexing scientific articles, and only according to the relatively general (for us) MESH keywords. A great deal of the material in BVQ such as meeting reports, book reviews, news, and editorial followup type material is therefore not NLM indexed. Index Binoculus also indexes scientific articles with more detailed and specific terms than MESH, facilitating your retrieval of information.]

Last year in the first issue of 2002, we updated and wrote here:

"Now in 2002, we enter yet another phase.

A combination of events has contributed:
1. This "mom and pop" operation, successful for 17 years, is finding it harder and harder to keep up with the latest advances in the use of computers, (no thanks to Bill [the fraud] Gates) and the new on line services provided by large publishers.
2. There have been in the last two years, several exciting medical problems for your editor, which have left him fortunately unimpaired but which have made him realize that he's not going to be around forever, and it is time to look for a permanent home for BV&SQ, while I am still able to do so.
3. The journal has enjoyed cooperative efforts of co-promotion with Swets & Zeitlinger, the Dutch publisher of Strabismus. Now they are interested in merging the two journals in the near future.

Keep being a subscriber, but keep tuned for future events!

(Continued on next page)
Update 2003: Well, it (2002) was an exciting year but it didn't turn out quite as we had intended or hoped. We have described some of those happenings in detail already in these pages [See BV&SQ 17(2):76 and 17(4):278]

1. Long time Charter Editorial Board member and [former] friend Carlos Souza-Dias, as outgoing Prexy of the ISA, cleverly blocked our well planned proposal for a combined BV&SQ and Strabismus to become the permanent ISA journal because he thought $49 a year (fantastic price I negotiated with S&Z) for one merged journal which combined two journals currently costing $84 + $126 = $210 per year - that this was a NOT a reasonable price for ISA members, who were already being taxed about $75 per person per year to pay for fellowships for non ISA members!

2. John Martin, the new head of Publisher Swets and Zeitlinger had so much trouble managing Huibert Simonsz, the Editor of his S&Z "Strabismus" Journal, that he changed his mind and upped (insisted upon) his requirement for personal total 100% Editorial control of the merged journal, in spite of the fact that nowhere in the scientific publishing world is this done by anyone. No M.D. was happy with that.

3. The S&Z Board, in spite of the fact that they would more than double or triple their profit by buying BV&SQ, reneged totally on previously discussed offers for BV&SQ. They limited their final offer initially to only one sixth and then finally to only one third of what they had offered in preliminary discussions. I had been warned of Dutch businessmen but this was the biggest fraud that this publisher has ever been subjected to by anyone. [P.S. except Bill Gates, of course]

Some unintended consequences of these events were enumerated in last issue's lead editorial. Chief among these is Editor Burt Kushner, after 17 years and 68 cases of his superb strabismus "Grand Rounds", deciding to move on to other major projects since he too has not yet found the fountain of youth, and after almost two decades wishes to move on to other projects. We plan to publish in a book, all 68 of these articles.

Fortunately your Editor in Chief's repaired mitral valve is doing OK, and his ventricular tachycardia has not returned, and his retinas remain attached, so we can continue on for the present as we were. Thanks to a peculiar accommodation of one of our variable annuity investments, when I do finally kick the bucket, there will be enough bread available to set up a foundation to support an independent BV&SQ permanently.

In the meantime we are refreshing the Editorial Board with some new and younger faces, whom we hope will help to see that the foundation does its job, forever.

Update BV&SQ 2005; 20(1): 4-6

Since the last printing of this history two years ago, with the help of Marcia Youngdahl, owner of our local print shop, we did get that book of Burt Kushner's 68 editions of his Grand Rounds published last year. We were all quite happy with the way it turned out. We did the best we could to copy the gorgeous cover style of Jean Paul Wayenborgh's History of Ophthalmology, but in a rich red rather than his royal blue.

Everything else continues unchanged in the lives of your FE and his publisher-orthoptist-wife. She put out Burt's book virtually single handed last year. [P.S. But who would have thought that Slack would continue publishing the JPOS, or that so many AAPOS members would be so willing to help them do so considering how poorly they treated the AAPOS and its membership for all those years? Who would have thought we could have not just one but FOUR scientific periodicals servicing our subspecialty — and surviving?]

Amazingly this is our twentieth volume and our twentieth year of publication. We plan to celebrate the completion of our twentieth year during the annual AAPOS meeting about this time next year, which will be held just five miles from our home and offices, up the road at the Keystone ski resort.

-PER

Update 2007 BV&SQ 22(1)

That 2006 AAPOS meeting next door was most successful but our 9500 foot altitude was not well tolerated by too many participants (see Dr. Mims III's report published in our pages in the Q2 summer issue page 102) so a repeat is not likely. There are, however, many good ski resorts available in the more comfortable 8000 or so foot range like our neighbor Vail.

At that meeting I found out that our recent myopia collaborator-contributor Michael Chiang was limiting his periodical subscriptions to those available on the internet. That did start us thinking about what you see culminating, thanks to a host of factors, including many advantages, in this first issue for 2007 conversion to an electronic internet version (see Editorial in this issue on following pages 15-16).

-PER

Revised and updated 2003; 18(1):4-6
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TEXT CONTENT: Manuscript material should be organized into the following parts in this order: ABSTRACT; INTRODUCTION (BACKGROUND AND PURPOSE OR PROBLEM); MATERIALS, SUBJECTS AND METHODS; RESULTS; DISCUSSION OF RESULTS; CONCLUSIONS (& recommendations) REFERENCES; TABLES; LEGENDS FOR FIGURES; FIGURES.

In the "Discussion of Results", do not introduce new reference material. Instead, we expect you to integrate YOUR NEW RESULTS into the current body of knowledge. Specifically: your results should be compared to results obtained by prior workers: Confirmations and agreements should be pointed out. But discordances also require enumeration, discussion, and explanation. Unique or unexpected results demand interpretation. The statistical significance* of results must be considered and their application should also be entertained.

REFERENCES: Order these numerically in sequence as they appear in the text. Indicate a reference number in the text with a full sized Arabic numeral enclosed in parentheses, i.e. (1). On the separate Reference page they should be numbered consecutively and typed double-spaced. Author's names and Journal titles should be abbreviated, without periods, as in Index Medicus. For journals punctuate in the following order: Author(s) last name Initials [et al] acceptable for more than 3; [colon] Article title with sub-title, if any.[period] IM Journal abbreviation [Bolded] Year; volume number in Arabic numerals: inclusive pages. Example. 1. Jones AB, Jones CD, Jones EF, et al: Results of Laser Surgery for Strabismus. J Outst Surg 1999; 2:301-304.

For book references: author, title, volume (if more than one) edition number (if other than the first), publisher, city and year. If the reference is a chapter in a book, the order changes as follows: the author of the chapter, the title of the chapter, "in" book title, volume, edition, editors, publisher, city, year, inclusive pages of the chapter. Authors are responsible for accuracy.

TABLES: Always "portrait" (< 7" W). NOT "landscape" configuration which requires undesirable sideways position. figures: PHOTOS, GRAPhICS, DRAWINGS

Electronic submission, email or on CD is usually acceptable. Standard Hard copy methods: Photo materials for halftones (photographs, photomicrographs, electron micrographs, roentgenograms) should be submitted cropped and unmounted. On the back of each print, affix a typed label with the figure number, an arrow and/or "top" indicating the top edge, and the last name of the first author. Line drawings, charts, and diagrams should be professionally prepared. For computer generated graphics, please submit originals, rather than photographic prints. Typed labels and lettering are not acceptable in graphics. Please ensure that lettering is large enough to be legible if and when reduced for publication.

Legends for Figures: typed double-spaced in consecutive order on a separate page following References. Start each with first author's name in parentheses. Indicate scale when appropriate. State clearly the point which the Figure is illustrating. Use arrows on photos liberally to identify and point out structures. (Assume the reader is not an expert like you are but rather an ignorant student.)

SOURCES, CREDITS, PERMITS

Quotations must be accurate and give full credit to the source. Brief properly credited quotes do not require permission of the original author or publisher ("fair use"). For large amounts of text or any figures previously published permission to quote and reproduce must be obtained by the submitting author: original copyright, the letters from the original author and publisher granting permission to reproduce the work must accompany your manuscript. Photo permits: if the subject can be recognized, i.e., any picture which contains more than just eyes and an unidentifiable bridge of the nose, written permission to publish the picture must be obtained from any subject over age 8 years old (and the parents if a minor under age 18) and submitted with it.

* Statistical Analysis of Results Mandatory. But give "exact" probability values (i.e., p = .06). Do not use relative p values (i.e.,p >> .05). The term "statistically significant", defined traditionally as a p < .05, is a totally arbitrary and unscientific term and should not be used (J Lab Clin Med 1988. 111:501). But do consider whether a result may be "clinically/medically significant".

rev 22.10/05 PER
D. BRIAN STIDHAM MEMORIAL LECTURESSHIP

LECTURE to be published annually in Binocular Vision and Strabismus Quarterly

Donations Sought to Fund Lectureship

To the Editor:

The Pediatric Ophthalmology community lost a great doctor last October 6, 2005, with the death by murder of D. Brian Stidham. I am attempting to create an endowed lectureship to remember Brian in our community and within pediatric ophthalmology, and wonder if I could ask you to consider helping in this regard. I know that your journal concentrates on strabismus and binocular vision, but could I interest you in publishing the "Stidham Lecture in Pediatric Ophthalmology and Strabismus" that will hopefully be given on a yearly basis? I would work with the presenter to make certain that a manuscript would be produced that would be of acceptable quality. Having a target journal for the presentation would be a great carrot to draw top speakers to Tucson on a yearly basis to give such a talk.

We have raised $14,000 towards a target of $50,000 endowment that would ensure that the lecture would be perpetuated. I am committed to continue fundraising until the goal is met. If Binocular Vision and Strabismus Quarterly would serve as the publisher of the named lecture, I feel certain we will be able to both attract top speakers and donors to remember Brian in the years ahead, and to provide a great lectureship in pediatric ophthalmology and strabismus to our professional community which would enjoy greater readership and distribution.

Joseph M. Miller, M.D., MPH
Head, Ophthalmology and Vision Science
University of Arizona, Tucson, Arizona

In reply:

We are honored to be asked and will most definitely be pleased to publish this lecture each year. We would encourage our readership to donate to this fund: Checks should be made payable to The University of Arizona Foundation with memo of "Stidham Endowment" and sent to Dr. Miller at U AZ, Ophthalmology, 655 N. Alvernon Way, Ste 108, Tucson AZ 85711.

ADVICE for authors submitting papers to Binocular Vision & Strabismus Quarterly©

1. READ & FOLLOW INSTRUCTIONS FOR AUTHORS! In addition:
2. READ & FOLLOW INSTRUCTIONS FOR AUTHORS! In addition:

Reviewing the literature: A proper review of the literature starts with a review of current and appropriate textbooks, especially the latest edition (currently the Sixth of von Noorden’s Binocular Vision and Ocular Motility by Mosby, and Duané’s loose-leaf text Clinical Ophthalmology. Anticipating a future requirement, it will only be to your credit now to specifically state what was included in your literature search, i.e., the topics or subjects and the sites searched. For any article submitted here that should include at a minimum, Index Medicus (Medline) from 1966 to the present, Index Binoculus Primus, 1985 to the present, and the Internet for the American Orthoptic Journal.

Acceptable TERMINOLOGY not acceptable

AHP Abnormal Head Posture face turn
head turn
chin up/down
head up/down
Head tilt
Fadenoperation
posterior fixation
suture
rethroequatorial myopexy
retroequatorial myopexy
hang back, hang
loose

Bielchowsky Head Tilt Test
three step test
Strabimology, ist
“Statistically significant”

exact p values

Re: "lost to followup" - Avoid this at all costs; First it raises the possibility that the patient had a (=) bad result or was otherwise so unhappy with their care that they never came back - or went elsewhere or went nowhere out of fear or dissatisfaction. If they are "lost followup" you just cannot refute the possibility that one those very unhappy thingsppened! Second it is inexcusable - medico-legally. Third: It reflects poorly on you as both a health care professional and as a scientist and Fourth: under the worse of circumstances suggests or indicates that you may discriminate against those of lower socio-economic status (research findings).

WRITING STYLE IS IMPORTANT TOO:

(Writing Style is Important Too) FROM INVESTOR'S BUSINESS DAILY NOV. 26, 1997 BY MOREY STETTNER

"Make Dry Data Come Alive in Your Reports ... tips on making your technical writing come alive:" 1. Remember that less is more. ... simplify your language and prune extra words. Eliminate jargon, and keep your sentences and paragraphs short. 'If you write in little bites, you break down lots of information for the reader so that it's easier to absorb,' said Carolyn Mulford, president of The Writing Coach ....

2. White in the active voice. ... For example, write 'When you review the data, you will note these trends'. Avoid saying 'These trends were noted upon a review of the data.' Another example: Write 'We will examine', not, 'This has been examined' ....

3. Insert 'talking subheads'; ... unbroken text can intimidate any reader, ... organize your writing in sections with each carrying an easy to understand subhead ... a talking subhead ... alerts the reader of what you're about to discuss ... for instance, instead of heading a section with 'Cost of Scanners' try 'Rising Cost of the Next Generation of Scanners'. ... subheads should average 7 words.

4. Run a test. ... ask someone in your audience group to read it.

TABLES: Don't forget the crowding phenomenon. It works in Tables too. We prefer spaces to lines to separate the items in a Table. You can also get more material within whatever size limits you may have, using spaces instead of lines, especially vertical lines. Horizontal lines are less of a sin.

-PER
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<td>Seattle</td>
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<td>Maria Schweers, CO  Tel: 515-964-7835 FAX: 515-964-7831 <a href="mailto:maschweers@mchsi.com">maschweers@mchsi.com</a></td>
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<td>Joanne Angle Tel: 240-221-2900 Fax: 240-221-0370 <a href="http://www.arvo.org">www.arvo.org</a></td>
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<td>Laurie Hahn-Parrott, CO, COT 3333 Burnet Ave. Pav 4-510 Cincinnati OH 45229-3039 Tel: 513-636-8168 FAX: 513-636-7911 <a href="mailto:lhparrott@yahoo.com">lhparrott@yahoo.com</a></td>
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<td><a href="mailto:kross@eyesite.ca">kross@eyesite.ca</a> <a href="http://www.eyesite.ca">www.eyesite.ca</a> Andrea Quan, TCOS President <a href="mailto:aquam@cw.bc.ca">aquam@cw.bc.ca</a></td>
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<td>AAO: FAX: 415-561-8575 <a href="mailto:customer_service@aao.org">customer_service@aao.org</a> AACO: Ron Biernacki, CO 518-262-2502 <a href="mailto:ronald.j.biernacki@vanderbilt.edu">ronald.j.biernacki@vanderbilt.edu</a></td>
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**Future AAPOS Meetings:**

- **2008**  Washington, D.C.  April 2-6, 2008
- **2009**  San Francisco, California  October 24-27, 2009
- **2010**  Chicago, Illinois  October 16-19, 2010
- **2011**  Orlando, Florida  November 5-8, 2011

**Future AAO/AACO Meetings:**

- **2008**  Atlanta, Georgia  November 8-11, 2008
- **2009**  San Francisco, California  October 24-27, 2009
- **2010**  Chicago, Illinois  October 16-19, 2010
- **2011**  Orlando, Florida  November 5-8, 2011

**Future ARVO Meetings:**

- **2008**  Fort Lauderdale, Florida  April 27-May 2, 2008
- **2009**  Fort Lauderdale, Florida  May 3-7, 2009

**World Ophthalmology Congress**

The name World Ophthalmology Congress was approved last year and will replace the former and traditional "International Congress of Ophthalmology". Therefore the 2006 World Ophthalmology Congress corresponds to the 30th edition of the ICO.

**Future Meeting Dates:**

- **2008**  China, Hong-Kong  June 28-July 2, 2008  www.woc2008hongkong.org
- **2010**  Berlin, Germany  June 6-10, 2010  www.woc2010.de (combined meeting with AAO)

**XI International Orthoptic Congress**

The XI International Orthoptic Congress will be held in Antwerp, Belgium, in May 2008. Start making plans now to attend.

Gill Roper-Hall has been selected to give the Burian Lecture.
For more information, visit: www.ioacongress2008.org
The official annual publication of the British and Irish Orthoptic Society, the Journal contains papers covering orthoptics, ocular motility, amblyopia, binocular vision, strabismus, related paediatric ophthalmology and neuro-ophthalmology.

The editorial board comprises leading British and Irish orthoptists and ophthalmologists.

Original articles for publication may be submitted to the Editor:
Dr Sarah Shea PhD DBO(D), Orthoptic Clinic, North West Wales NHS Trust, Tŷ Bydny Gwynedd, Bangor, North Wales LL57 2PW United Kingdom

American Orthoptic Journal
is your main focus for these fields!

- Ophthalmology • Pediatric Ophthalmology
- Neuro-Ophthalmology • Strabismus
- Amblyopia

For ophthalmologists, orthoptists, and related ophthalmic personnel, this professional resource focuses on amblyopia and strabismus. AOJ presents: information from papers presented at North American regional and national meetings
- latest clinical research articles • abstracts from publications around the world including: German, French, and British journals • and book reviews.

Through the journal, members of the ophthalmologic and orthoptic communities can keep abreast of current clinical practice and research in the fields of ocular motility and amblyopia.

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MEETING REPORT !!!


William Anninger, M.D. Appointed

The Children's Hospital of Philadelphia is pleased to announce the appointment of William Anninger, MD, as attending physician, Division of Ophthalmology. Dr. Anninger has been educated at the University of Michigan, Dartmouth Medical School, interning at Cambridge City Hospital of Harvard University, completing his ophthalmology residency at the William Havener Eye Institute, at Ohio State University. He completed his pediatric ophthalmology fellowship at the Children's Hospital in Philadelphia. He can be reached at the main campus at 215-590-2791.

RESEARCH OPPORTUNITIES

Research Funds Available

The Blind Childrens Center (BCC) is again pleased to announce the availability of funding to support research to gain better understanding of visual impairment in children from birth to 5 years of age. One year grants of up to $15,000 will be awarded. For questions and application forms, contact Midge Horton, Executive Director, Blind Childrens Center, 4120 Marathon St, Los Angeles CA 90029. Tel: 323-664-2153 ext. 326.

The Contact Lens Association of Ophthalmologists has announced the availability of 3 grants, of up to $10,000 each, to be awarded to provide support for research specifically concerned with issues directly related to contact lens science and/or ocular anterior segment science. Grant applications may be obtained by visiting the CLAO Home Page at www.clao.org or by calling the office at 877-501-3937.

Study Recruitment

Photorefractive Keratectomy (PRK) for Ametropia in Children

Evelyn A. Paysse, MD of the Baylor College of Medicine, Texas Children's Hospital, and colleagues are recruiting patients to participate in a funded study for children with bilateral ametropic amblyopia who have been non-compliant with traditional treatment. The study involves using PRK to fully or partially neutralize the refractive error and thereafter following the children for a one year period. They will also be collaborating with the developmental pediatric physicians to determine if changes in visual outcome affected the development of children with amblyopia. If you have children in your practice between the ages of 2-7 years old with a bilateral high myopic ametropia of at least 7 D, bilateral high hyperopic ametropia of at least 4 D, or bilateral high astigmatic ametropia of at least 3.5 D, who are non-compliant with traditional treatment options, please consider enrolling them in this study. Contact Dr. Paysse at 832-822-3237 or her research coordinator, Maria Castanes at 832-822-3257.

Practice Opportunities

Fresno, California: Large MD/OD practice. State of the art facility and satellite office. Affiliated with Children's Hospital of Central California. $10,000 signing bonus, bonus plan, benefit and relocation package. Fax resume to the human resource department at 559-256-8504 or contact Scott Bridgman, CEO at 559-256-8500. Visit their website at eyeqvc.com

Albuquerque, New Mexico: Children's Eye Care of New Mexico. Two hospital affiliations. Major contributor/participant for PEDIG studies. A division of Goldblum Family Eye Care Center. Contact: Todd Goldblum, MD, 303 Mulberry NE, Suite D, Albuquerque NM 87106. Fax: 505-842-0650. email: todd@goldblumeye.com

Public Safety Hazards

from Academy Express February 8, 2007. Is A Complete Ban of Cell Phones in Ophthalmology Departments Necessary? British researchers placed cell phones in contact with 23 electronic ophthalmic devices, as well as at distance of 3 m, 1 m and 30 cm away. No device showed any interruption or cessation of function.
EDITORIAL: Here We Go, Charging Full Throttle and Headlong into the Internet.  
Binocular Vision and Strabismus Quarterly 
Stops the Presses! All But Totally, and Re-establishes Its Old Website at BINOCULARVISION.NET for Continuing E-Publication of this Now E-Periodical

It has been a monstrous amount of work over the past four or five months, about a thousand hours of my time, primarily because we have had to finally give up our addiction to old WordPerfect 5.0 word processing and learn Gates’ (biggest crook in world history) Hated Word since WP 5.0 is not convertible to Word. In fact, WP 5.0 isn’t really convertible directly even to WordPerfect 10 or anything beyond WordPerfect 5.5. WordPerfect 10.0 will at least accept and partially convert our 5.0 files to 10. And WP 10 does still have its original great “Reveal Codes” feature which is vastly superior to the minimal code information provided by Bill in Hated Word.

Since yours truly does most of the layout work, it was he who had to learn all this new stuff. It has taken me reading, working, help-begging, and the infinite trial and error trial and error at the keyboard that is so unavoidable, somewhere about twenty weeks of 50-60 hours work each to learn enough about Hated Word and WordPerfect 10, to process and finish the manuscripts for this first issue of our twenty-second year.

BV&SQ will be available for all subscribers at Binocularvision.net by the time you read this. In fact, you may have already been notified by e-mail with a link directly to that website, and may have already read this issue. We have tried to maintain the traditional appearance of the old yellow printed BV&SQ with which you are all familiar.

We have adopted a somewhat larger type font because there is some unpredictable loss of resolution when you put the typeset pages through the process of becoming pdf’s (“portable document files or format”) which is de rigueur for the internet so that everybody can read them (with the help of the free Adobe reader software, of course). But we have no idea exactly what equipment each subscriber has to view each issue on the web, so for starters, better too big than too small.

As a subscriber, there is nothing more for you to do than going to the website, signing in and viewing the pages of each issue. The subscription rates are unchanged. If you also paid extra for airmail, that will be refunded to you. Instead of receiving a hard copy of BV&SQ by post, you will get an email notifying you that the next issue is available on our website and the email will contain a hot link to the website, so that all you will have to do when you get there is sign in, then select whatever parts of that issue you want to read and/or download and make hard copies at no fee. There will be no time limits on your use of any material on the website. We will archive, on the website, all issues from now on and as a subscriber you will have permanent 24/7/52 access to all issues from here on. And in the future we plan to also
archive on the website all previous 84 issues since 1985.

Now for those who need or want, in addition, an old fashioned hard copy of the issues, we hope to “hand-make” some of these because we would like to provide all contributors with at least one “original” hard copy from which they can make original-like copies for their fans, chairman, bosses, and promotion committees. But since we will not be printing hundreds of copies using the printing press, such copies will be expensive, for us, and for you. We are estimating about $47 per issue hard copy (which is coincidentally for now the same that it will cost non-subscribers to purchase on the website just the right to read and download a whole issue for one week. Therefore, nonsubscribers will have to pay $188 per year to view BV&SQ one issue at a time for a week at a time. As a subscriber, you are paying a maximum of only $84 per year (and as little as $64 a year for 3 year subscribers) for the same right. In addition to paying $104 to $124 less per year, you will have permanent anytime access to every issue.

In This Issue

This is sort of an amblyopia issue in that the two major original scientific research articles are on amblyopia, and similarly consider aspects of the final results of our conventional current treatment.


This is huge and outstanding piece of work and Dr. Dr. (I do love that continental doubling way of recognizing PhD’s) Johnson sent us an abridged version of his original AOS paper. One might have expected his remarkable discovery if you were as familiar with the amblyopia literature as he is. And he shares that knowledge with us in the extensive review of the amblyopia literature within his Discussion. It is a superb review. Unilateral amblyopia is a disease of a bilaterally represented part of the brain and so is truly always really a bilateral disease!


These orthoptist researchers were as dismayed as the rest of us when penny pinchers in Britain tried to destroy our intentions to find and treat amblyopia in children (see their reference 1) as unworthy. But they did something about it. They did some research which well refutes those obsessive-compulsive “evidence-based medicine” Brits. They were ready to destroy our efforts as medical professionals, doctors, and orthoptists and technicians totally just because there were insufficient perfect randomized controlled outcome studies. They are really NUTS! And that goes for all you “authorities”, editors and such out there who share that obsession. That’s not rational science or medicine.


We must commend Dr. Ken Wright for his enthusiastic creation of these many strabology /pediatric ophthalmology meetings. They do recreate a happier time for most of us, a time when there were fewer of us to attend our meetings, and it was easier to contact older original friends from training times, always somehow reassuring. A three-peat is scheduled for the 2007 NOLA AAO

Both because of the length of Dr. Dr. Johnson’s major and important thesis, and to simplify creation of our first e-issue, there is no section on abstracts, no Hyde Park Editor’s soapbox-blog. But rest assured, the second issue this year will see those features restored. And you will see it soon -PER.
Relative Scotomata in the "Normal" Eye of Functionally Amblyopic Patients.  
A Scanning Laser Ophthalmoscope (SLO) Microperimetric Study

DAVID A. JOHNSON, M.D., Ph.D.  
from Eye Associates of Wilmington, Wilmington, North Carolina

ABSTRACT: **Purpose:** To evaluate amblyopic patients with scanning laser ophthalmoscope (SLO) microperimetry to determine whether SLO assessment and data might provide useful information in our understanding of amblyopia and determine its utility in the evaluation of amblyopic patients.

**Methods:** In this retrospective, selected for SLO testing case series, clinical data of forty-six patients with amblyopia were reviewed after completion of treatment for anisometropic or strabismic amblyopia. Ten ophthalmologically age-matched normal patients served as controls. All patients were tested with the SLO, specifically evaluating for the presence of macular scotomata. SLO findings were assessed within each group and between groups.

**Results:** A macular scotoma was found in the amblyopic eye of 25 of 26 anisometropic amblyopic patients and all 20 strabismic amblyopia patients. Twenty of the 26 patients with anisometropic amblyopia also had a relative scotoma in the non-amblyopic “normal” contralateral eye. All 20 patients with strabismic amblyopia also had a non-amblyopic “normal” contralateral eye scotoma. None of the normal control patients had a scotoma in either eye. Several ocular and binocular clinical features were correlated to these scotoma findings within and between groups.

**Conclusion:** The SLO proved useful for the assessment of some features of amblyopia. A scotoma was identified not only in the amblyopic eye of all but one amblyopic patient, as expected, but also in almost all of the fellow non-amblyopic, presumed "normal" contralateral eyes, and in spite of treatment normalization of visual acuity and stereoacuity in several cases. Thus, the ocular and binocular pathological effects of unilateral functional amby-opia are not limited to the amblyopic eye but may also be seen, to a subclinical degree, by SLO microperimetry in the supposedly normal contralateral eye as well as in the apparently successfully treated previously amblyopic eye.
INTRODUCTION

Amblyopia is a common cause of visual loss, despite increasing understanding of its causes and mechanisms. It is defined (1) as a "unilateral or bilateral decrease of visual acuity caused by form vision deprivation and/or abnormal binocular interaction for which no organic causes can be detected by the physical examination of the eye and which,... is reversible by therapy[2]." Amblyopia is estimated to affect 1-4% of children (2,3) and approximately 2.9% of adults (4). Its pathophysiology stems from early abnormal binocular interactions (usually from strabismus) or form vision deprivation in one eye (from uncorrected anisometropia) or, less commonly, both eyes (uncorrected high hyperopia). Not infrequently, multiple factors are involved in the same patient.

Numerous approaches have been employed in the clinical and research study of amblyopia. Visual acuity measurement in the clinical setting usually is by age-appropriate subjective testing, methods such as Snellen charts, Allen cards, Tumbling E game, etc. Pre-verbal children require forced choice preferential looking tests (Teller cards), determination of fixation preference when strabismic, or induced tropia testing when eyes are grossly aligned. Evaluation may include measurement of contrast sensitivity and subjective tests of binocular cooperation, such as Worth 4 Dot Test and stereopsis. More recently, research methods have employed newer techniques, such as functional magnetic resonance imaging (fMRI) (5) or positron emission tomography (PET scanning) (6) in the investigation of the cortical neuro-physiologic mechanisms underlying amblyopia. Histological evaluation of visual neuronal pathways in visually deprived animals and humans has been studied as well (7-11).

The advent of the scanning laser ophthalmoscope (SLO) has brought another tool into the armamentarium of the eye clinician. The SLO has been used to advance the evaluation of the optic discs in glaucoma, by providing greatly enlarged, quantitative, three-dimensional imaging and analysis of the disc surface (12, 13). Other advantages of the SLO include micro-perimetry, where it has been used to enhance evaluation of point of fixation and fixation stability in conditions such as macular dystrophies (14), idiopathic macular holes (15), age-related macular degeneration (16,17), and pre- and postoperative visual function after retinal surgery (18). Another area in which the SLO has been particularly valuable is in low vision rehabilitation, where micro-perimetry aids not only in the localization of a patient's preferred retinal locus of fixation, but also as a device to assist rehabilitation of low vision patients in fixation stability and vision optimization (19-21).

The purpose of this study was to use the SLO as a microperimetry device to assess macular stimulation threshold, and the presence and patterns of scotomata in the eyes of post-treatment amblyopic and normal control patients. Further analysis was used to find distinctions between different etiologies of amblyopia and to seek identifiable characteristics of patients that correlate with their clinical response, or lack thereof.

SUBJECTS and METHODS

Patients:

Forty-six patients with previously treated amblyopia were selected from their prior treatment records and studied by SLO exam. Patients included those with strabismic or anisometropic amblyopia. Ten ophthalmologically normal patients served as controls. Human studies committee and institutional review board approval was obtained and informed consent granted by each subject's parents. HIPAA regulations were enforced.

Amblyopia was defined as meeting one of these three criterions:
1. in pre-verbal children, a strong fixation preference for one eye over the other if strabismic or by induced tropia testing (vertical prism) if child was orthotropic.
2. an interocular visual acuity difference of ≥2 lines using projected pictures in age-appropriate children on a B-VAT vision measuring instrument (Mentor O & O, Norwell MA) at 20 feet testing distance; or
3. an interocular difference of ≥2 logMAR lines at distance visual acuity testing with the B-VAT, using either Snellen letters or
HOTV letters with surround bars at a 20 foot (6M) testing distance.

Children who were pre-verbal at the onset of treatment were included in this study only if followup was sufficiently long that a final visual acuity could be determined by criteria number 3 above.

To divide the amblyopic patients into two manageable groups for comparison and analysis, strabismic amblyopia was defined as that occurring in strabismus of at least ten prism diopters, without anisometropia. If a strabismus was less than ten prism diopters and there was clinically significant anisometropia, these patients were placed in the group of anisometropic amblyopia. Most of these patients were in fact orthotropic. Clinically significant anisometropia was defined as an interocular spherical refractive error difference of 1.0 diopter or greater or an astigmatic difference of 1.50 diopters or greater. For purposes of data reporting, the refractive error difference between eyes was regularly expressed as the spherical equivalent.

Details of prior treatment of amblyopia: in all cases, a patient was not diagnosed as having amblyopia, nor was treatment started, unless the defined difference in interocular visual acuity persisted after at least 4 weeks of spectacle correction of refractive errors, when appropriate. Need for spectacles was based upon cycloplegic refraction and, when age-appropriate, a cycloplegic refraction.

All patients included in the study also needed to be free of confounding coexisting ocular and binocular disorders that might be responsible for decreased visual acuity or coexisting medical problems that might limit reliability of visual acuity measurements and any testing measures.

The normal control patients selected, in the same age range as the amblyopic patients, were required to demonstrate 20/20 best corrected visual acuity in each eye, normal binocular alignment, 40 seconds of arc stereacuity and normal ocular movements and anatomy.

Amblyopia was treated conventionally either by occlusion (by adhesive patching) or atropine (1% eye drops) penalization of the normal contralateral eye. Atropine penalization was instituted only in patients whose visual acuity in the amblyopic eye was 20/100 or better.

Some patients began amblyopia treatment while still pre-verbal and, therefore, in them, initial acuity was assessed by fixation preference if strabismic, or if not, by induced tropia testing, if orthotropic. However, interim and final visual acuity was measured in all patients at 20 feet (6 M) using age-appropriate testing, including projected pictures, HOTV optotypes (with surround bars) or Snellen letters. All stimuli were presented on a calibrated B-VAT. All Snellen visual acuities were converted to LogMAR format for proper statistical analysis (22).

Patients were excluded if they had undergone amblyopia therapy prior to initial evaluation by the author. Patients were included only after having completed a course of amblyopia therapy by the author and were felt to have reached either (1) equal visual acuity in the two eyes or (2) a less than equal but improved and stable acuity in the amblyopic eye which had remained stable after amblyopia treatment had been discontinued for at least 3 months.

Because performance of the SLO testing involves considerable cooperation, patients were excluded from the study if they could not cooperate and follow instructions while maintaining steady fixation on a target at the slit lamp biomicroscope, which simulates the subject-patient cooperation necessary to perform the SLO measurements.

Ophthalmologic measurements obtained included visual acuity, binocular alignment, stereopsis at near using the Titmus stereacuity test, the 4 diopter base-out prism test, Worth 4 Dot test, slit lamp examination, dilated funduspic examination and cycloplegic refraction. Amblyopia data obtained included best corrected visual acuity in both eyes at beginning of treatment and end of
treatment, age at onset of treatment, method of amblyopia treatment, treatment duration, need for re-treatment, age at SLO testing and all follow-up time from the onset of treatment to last follow-up.

**Microperimetry:**

Microperimetry was performed with a scanning laser ophthalmoscope (Rodenstock Instruments GmbH, Model 101, Ottobrun-Riemerling, Germany). This SLO is a Class 1 Laser appliance and complies with laser protection regulations DIN-VDE 0837 and IEC 825. This lowest laser classification is achieved because the laser beam is always constantly scanning the retina and never remains static on the same spot. Safety shutdown security is in place for failure of the scanning mechanism or limits on the duration of examination.

SLO microperimetry is similar to conventional perimetry in that it is a psychophysical test. That is, a stimulus is presented at a point remote from fixation and the patient is to respond if the stimulus is appreciated or recognized. Except for the normal control patients, all study subject-patients tested had clinically-defined amblyopia. All test points were applied in the macular region. The testing strategy is described below. The scanning laser ophthalmoscopic microperimetry was performed by a low vision specialist who was masked to patient diagnosis.

**Definitions:**

The microperimetric stimulus Threshold was defined as the least intense stimulus which elicited a reliable recognition response. The threshold for a given eye may or may not be in the foveal area. During testing, threshold was defined as the stimulus level at which the patient recognized approximately 50% of the presented stimuli. Testing was begun at a level determined empirically to be that recognized by most patients. That intensity was reduced until the patient could no longer recognize a stimulus and then the stimulus intensity was increased until approximately 50% of delivered stimuli were recognized. The location of greatest sensitivity and the threshold level in any given eye may be quite different in any individual eye from that point in the fellow eye. This point may be centered on the fovea. The point of greatest retinal sensitivity in an eye with a relative scotoma may be more distant from the fovea than the most sensitive point in the fellow presumed normal eye. It follows that the entire macular area of an eye may have a higher threshold than that of the fellow eye and within itself contain an absolute or relative scotoma.

An absolute scotoma was defined as a group of contiguous points in the macular area where the brightest of stimuli failed to elicit a recognition response. This corresponded to stimuli at 0 dB which was the brightest stimulus deliverable.

A relative scotoma was defined as tested points in a definable contiguous area in the macula where a brighter stimulus was required to elicit recognition than the intensity required for recognition in areas bordering it on all sides. That is, points outside the scotomatous area more distal to the fovea were more sensitive (required a less bright stimulus) than were points closer to the fovea.

Relative scotomata differ in "density"; i.e., the level of stimulus intensity (dB) required for recognition. In most patients with a relative scotoma, the sensitivity to recognition was uniform throughout the scotomatous region and a second relative scotoma was not identified. However, there were patients who demonstrated both an absolute scotoma and a surrounding relative scotoma. In those few patients, the scotoma area was considered to be that encompassed by the relative scotoma including the absolute scotoma.

**Testing:**

In SLO microperimetry, the retina is viewed at all times during the testing by way of an infrared laser (780 nm), at a background intensity of 10 candela/m² which is barely perceptible by the patient.

The examiner monitors the patient's macula as a gray-scale image in real time on a computer monitor (see Figure 1, next page, below, or right).
First, the patient is instructed to fixate on an illuminated cross 1.0 degree in size presented in the center of the field. The fixation cross is also visible as projected on the retina to the examiner on the computer monitor. Per the instruction, it is assumed that the localization of the fixation cross represents the patient’s preferred retinal locus of normal fixation. The examiner can also visualize whether this locus coincides with the anatomic foveal area as it should.

Now, a reference point on the retina (for example, the intersection of two prominent blood vessels) is chosen and set to allow compensation for unintended or undesired patient eye movements. If fixation locus slips or changes, accurate centration of the macular image can be maintained and the eccentric stimulus test point accurately registered (23). Thus, the examiner can assess, by direct and continuous real-time observation, the patient’s stability and locus of central or best fixation. As a result, a stimulus was not presented unless the patient was fixating properly. Hence, the concept of “fixation loss”, as is used as a measure of reliability in automated perimetry, does not apply in SLO microperimetry in that NO stimuli were presented if the patient was not properly centrally or maximally fixating.

The stimulus was presented directly on the retina by a Helium-Neon (HeNe) laser (545 nm). A stimulus with a diameter of 10 minutes of arc (=50 um diameter) was used. The stimulus could
be regulated and graded in intensity and was presented for 100 ms, thus generating a static, rather than kinetic, perimetry assessment. The instrument's stimulus intensity range is from a brightest possible intensity of 0 dB to the dimmest stimulus of 40 dB (a range equivalent relatively to 10 to the 40th power) and is adjustable in steps of 1 dB (10X difference for each dB; a 2 dB difference =100X). Patients were allowed 2.0 seconds to respond to each stimulus. When the stimulus was recognized, the patient pressed a toggle switch to register their perception.

When a positive response was made, or 2.0 seconds had transpired, the retinal image was frozen and the stimulus data point was registered on the macular image in the precise location where it was delivered. The previously designated anatomical reference point then allowed each stimulus to be marked on the overlay in its exact location relative to other stimuli.

On the SLO images, the perimetry stimuli presented are shown on the retinal image in true relative size and are color-coded for relative intensity. Stimuli are also, in addition, icon shape-coded to represent whether the stimulus was detected or not detected mapping the scotoma. Filled circles represent positively-recognized test points, open triangle a missed (unrecognized) test point.

Initial testing was begun with a 19 dB stimulus, which was an intensity empirically determined to be detectable by most subjects. The right eye was always tested first. The stimulus was first presented in close proximity to the fixation cross. Several points in this area were tested to determine whether the patient responded positively or failed to respond. If the patient failed to respond, the test area was expanded centrifugally to determine if there was any macular area in which the stimulus was detected. That is, testing addressed whether the patient responded to a 19 dB stimulus in a macular area more distal from the central macula (or fovea). If no areas of greater sensitivity were identified, the stimulus intensity was increased and the above strategy repeated.

If positive responses were noted in an area not at the point of fixation, the stimulus intensity was reduced to determine threshold at that location (see definition above). This, point then, represented a scotoma border. Numerous points were then tested in and out of this presumed scotoma, to define its borders and size. This process was continued until the scotoma could be mapped and outlined as accurately as possible, much like scotoma mapping is performed with Goldmann kinetic perimetry.

If a definable scotomatous region could be so identified, the stimulus intensity was increased within the scotoma borders until recognition, to determine the "depth" of the scotoma. That is, the intensity of a stimulus necessary to evoke a response within the scotoma was determined. This stimulus intensity was then used to "re-map" the scotoma borders. Although uncommon, it

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**Figure 2 see next page** (Johnson DA): SLO images of a control patient. The patient was instructed to fixate on an illuminated central cross. A reference marker (red cross) was marked at a prominent vessel crossing to allow all test points to be recorded in precise register on the image. After determining threshold sensitivity, the macula was scanned for scotomatous area. Scanning of the right eye (top) was started at 19 dB (yellow). When no scotoma was identified more peripherally, stimulus intensity was lowered to determine threshold and testing for scotomatous areas repeated. Based upon the threshold determined for the right eye, scanning of the left eye (bottom) was started at lower stimulus intensity (dark green). Stimulus intensity is greater at lower numerical values (red) and lessens in intensity as numerical values increase (brown, yellow, green, blue). Each colored bar represents a 5 dB range but stimuli could be changed in 1.0 dB steps.
was possible that a relative scotoma of greater "depth" might lie within a relative scotoma of lesser "depth" or an absolute scotoma might lie within a relative scotoma.

If there was a positive response to a 19 dB stimulus centrally, several points were tested more distally to determine if this level of sensitivity was uniform throughout the macula. Then stimulus intensity was decreased and the localization method described above was repeated. Such testing, as the stimulus intensity was reduced, might reveal a relative scotoma, but one requiring a lesser intense stimulus to discover and map than those that might be uncovered with the original 19 dB stimulus. This was done until the subject failed to recognize any stimuli presented. The stimulus intensity was then gradually increased until an approximately 50% positive response rate was reached. This was then determined to be the subject’s threshold, as defined above. This method was used to examine each quadrant unless scotoma mapping had already encompassed each quadrant.

Using the above strategy, the number of points necessary to thoroughly test a patient varied widely among patients, depending on the presence or absence of a scotoma, its borders and depth. For example, a subject in which a scotoma was not found could be tested for threshold and the macula scanned for sensitivity in a much shorter time than a patient with a complex scotoma shape and size. Measurements in each patient typically took 5-10 minutes per eye.

Retesting of missed points was performed to determine reliability. If retesting confirmed the initial finding, the examination continued. If not, further testing at that point was performed for accuracy in terms of scotoma size and stimulus intensity required for recognition. Because the test points are applied while viewing a real-time image of the macula, precision of retest points was very high.

However, such a high magnification view demonstrated that "steady" fixation does not mean "motionless" fixation when viewed at the microscopic level. Using anatomical landmarks, reapplication of a stimulus was typically very precise and within the range of no more than one-half stimulus width (one half of the standard stimulus diameter.)

The mapping of scotomata thresholds, size and depth, could be done with stimulus variations of 1.0 dB. However, the final SLO instrument printout only presents color-coded data points over a span of 5.0 dB; two "same" color points on the printout could be up to 5.0 dB different in intensity. Hence, scotoma boundaries were mapped and recorded as to stimulus intensity as the SLO was being performed, and not strictly from printed data, so as to allow as accurate a mapping as possible (see legend, to Figure 2 on prior pages).

The retinal images, with scotomata mapped, were digitally scanned and converted to bit-map form. The area was measured using AutoDesk Architectural Desktop 3.3 software (Autodesk, Inc., San Rafael, CA). Data were expressed as arc-min² based on a projected calibration image from the SLO.

Statistical Methods:
Data is presented as mean ± standard deviation. Data analysis was performed using Minitab Release 14 statistical software (Minitab, Inc., State College, PA). Comparisons between eyes of individual patients and comparisons in the same eye pre- and post-treatment were made using the paired t-test. Comparisons between groups were analyzed with the two sample t-test. Correlations between variables were assessed with the Pearson correlation coefficient test.

Statistical interpretations of these statistical test results were based on the conventional traditional definition of "statistical significance" being equal to a probability value (for the probability of rejecting the null hypothesis) equal or less than 0.05 (indicating the probability of the result not being due only to chance of less than 0.05 or one in 20), an entirely empirical value. If this probability is less than 0.05 or 20;1, say for example only...
0.06 or about 17:1, then that comparison of different items is said to be NOT "statistically significant" and is then, by convention and tradition, discarded as being NOT different from chance alone - rather, it could be due to chance alone and is therefore considered not due to some real-world factor or cause (being investigated). Statistical Significance so defined is here-after indicated by quotation marks as in "Statistical Significance" or "Statistically Significant(ly)"

RESULTS

Demographics:
A total of 56 patients were included in this study (Table 1). Ten were normal control patients, 46 were "experimental" subjects. These 46 patients comprised two groups, based upon diagnosis of amblyopia etiology type: anisometropic amblyopia (n=26) and strabismic amblyopia (n=20) (see Table 1, below). Twenty-five were male; thirty-one were female. Ages at time of SLO testing ranged from 66 to 146 months with a mean of 104.1 ±20.2 months.

Normal Control Subjects:
Ten patients (7 male, 3 female) served as normal control patients who underwent SLO testing (Table 1). Each of these subject-patients had normal: bilateral best corrected visual acuity, binocular alignment, binocularity, stereopsis, and normal anatomy. None had any prior ophthalmologic intervention other than spectacle wear for physiologic myopia in three patients. All were healthy with no confounding medical problems that might affect performance of the clinical or SLO examination.

The mean age of the normal control group at time of SLO testing was 110.0 ±25.1 months, which was not "statistically significantly" different from

TABLE I

<table>
<thead>
<tr>
<th>DEMOGRAPHIC AND TREATMENT CHARACTERISTICS OF ANISOMETROPIC OR STRABISMIC AMBLYOPIA</th>
<th>PATIENTS AND CONTROL PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ANISOMETROPIA</td>
</tr>
<tr>
<td>N</td>
<td>26</td>
</tr>
<tr>
<td>MALE:FEMALE</td>
<td>9:17</td>
</tr>
<tr>
<td>AGE START (months)</td>
<td>66.5 ± 22.2</td>
</tr>
<tr>
<td>FOLLOW-UP (months)</td>
<td>47.8 ± 26.4</td>
</tr>
<tr>
<td>AGE SLO (months)</td>
<td>104.0 ± 19.1</td>
</tr>
<tr>
<td>AMBLYOPIC EYE OD:OS</td>
<td>10:16</td>
</tr>
<tr>
<td>TREATMENT DURATION (months)</td>
<td>20.5 ± 12.6</td>
</tr>
</tbody>
</table>

Values expressed as mean ± standard deviation; NA, not applicable.
the 26 anisometropic amblyopia patients tested (p=0.51, two sample t-test) or the 20 strabismic amblyopia patients (p=0.35, two sample t-test).

Using the SLO, threshold was determined and the central macular area studied for scotomatous regions. Threshold sensitivity at the SLO was 26.0 ±3.9 dB, with a range of 18-31 dB. No scotomatos areas were found in either eye of any of the 10 control patients. The SLO images of a normal control patient are seen in Figure 2.

**Anisometropic Amblyopia Patients**

Twenty-six patients (9 male, 17 female) with anisometropic amblyopia were treated for amblyopia and tested with the SLO (see prior Table I). Of these 26, fully 20 were in fact orthotropic. The remaining 6 had small angle strabismic deviations, all under 10 prism diopters but also clinically significant anisometropia. The mean age at which amblyopia was diagnosed and treatment begun was 66.5 ±22.2 months, with a range of 24.0-113.0 months. Followup averaged 47.8 ±26.4 months (range 8-98 months). Mean age at SLO testing was 104.0 ±19.1 months with a range of 66-142 months. The right eye was the amblyopic eye in 10 patients and the left eye was in 16 patients. The mean difference in the refractive error between the amblyopic eye and the fellow eye, expressed as spherical equivalent, was 2.68 ±1.76 diopters.

The mean visual acuity at the start of treatment in the amblyopic eye was LogMAR 0.65 ±0.27 (approximate Snellen equivalent, 20/90, see Table II, next page) and, in the fellow, non-amblyopic eye, was LogMAR 0.10 ±0.12 (Snellen equivalent, 20/25). The mean acuity in the amblyopic eye after treatment was LogMAR 0.30 ±0.23 (Snellen equivalent, 20/40), reflecting an improvement of 3.51 ±2.29 logMAR lines, on average (range 0.9 lines; p=0.000, paired t-test, see Table II). Acuity in the non-amblyopic eye post-treatment was LogMAR 0.02 ±0.04 (Snellen equivalent, 20/21), with a change of 0.84 ±1.35 lines from pre-treatment (range 0.0-4.77 lines). This was also a "statistically significant" improvement (p= 0.002, paired t-test), although the average improvement in visual acuity was less than one LogMAR line.

The mean pre-treatment acuity difference between the amblyopic eye and the fellow eye was 5.53 ±2.41 LogMAR lines (range 2.0-11.0 lines) which improved to a mean post-treatment interocular visual acuity difference of 2.86 ±2.26 lines (range 0.0-9.0 lines, see Table II). This improvement/change/difference is "statistically significantly" different (p=.000, paired t-test), reflecting an average improvement of interocular visual acuity difference of 2.71 ±2.06 LogMAR lines.

Eighteen of these 26 patients had measurable stereopsis. Six of these patients had a stereoacuity of 50 seconds of arc or better.

Threshold sensitivity averaged 17.8 ±3.9 dB (range 9-24 dB) in the amblyopic eye and 22.5 ±2.8 dB (range 17-27 dB) in the non-amblyopic eye of these anisometropic patients (see Table II, and Figure 3, below). (Recall that a lower
**Figure 3, prior page (Johnson DA):** Boxplots of SLO threshold sensitivity in non-amblyopic (left) and amblyopic (right) eyes of patients with anisometropic amblyopia. The shaded box represents the middle 50% of observations with the horizontal line in the shaded area indicating the **median** data value. The lines extending vertically from the box extend to indicate the **range**: lowest and highest values among the data.

**TABLE II**

**VISUAL ACUITY DATA AND SCANNING LASER OPHTHALMOSCOPE CHARACTERISTICS OF ANISOMETROPIC AND STRABISMIC AMBLYOPIA PATIENTS**

<table>
<thead>
<tr>
<th>Number of Patients</th>
<th>Anisometropia</th>
<th>Strabismus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>26</td>
<td>20</td>
</tr>
</tbody>
</table>

**Visual Acuity (LogMAR) (Snellen Equivalent)**

<table>
<thead>
<tr>
<th></th>
<th>Amblyopic eye pretreatment</th>
<th>Amblyopic eye post-treatment</th>
<th>Non-amblyopic eye pre-treatment</th>
<th>Non-amblyopic eye post-treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.65 ± 0.27 (20/90)</td>
<td>0.30 ± 0.23 (20/40)</td>
<td>0.10 ± 0.12 (20/25)</td>
<td>0.02 ± 0.04 (20/21)</td>
</tr>
<tr>
<td></td>
<td>0.48 ± 0.15 (20/60)</td>
<td>0.25 ± 0.13 (20/36)</td>
<td>0.10 ± 0.08 (20/25)</td>
<td>0.06 ± 0.05 (20/23)</td>
</tr>
</tbody>
</table>

**Interocular Acuity Difference (LogMAR lines)**

<table>
<thead>
<tr>
<th></th>
<th>Pre-treatment 5.53 ± 2.41</th>
<th>Post-treatment 2.86 ± 2.26</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3.81 ± 1.47</td>
<td>1.97 ± 1.24</td>
</tr>
</tbody>
</table>

**LogMAR Lines Improved**

<table>
<thead>
<tr>
<th></th>
<th>Amblyopic eye 3.51 ± 2.29</th>
<th>Amblyopic eye 2.35 ± 1.81</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-amblyopic eye 0.84 ± 1.35</td>
<td>Non-amblyopic eye 0.50 ± 0.70</td>
</tr>
<tr>
<td>Threshold (dB)</td>
<td>Amblyopic eye 17.8 ± 3.9</td>
<td>Amblyopic eye 17.3 ± 5.8</td>
</tr>
<tr>
<td></td>
<td>Non-amblyopic eye 22.5 ± 2.8</td>
<td>Non-amblyopic eye 21.6 ± 3.3</td>
</tr>
</tbody>
</table>

**Scotoma Area (arc-min²)**

<table>
<thead>
<tr>
<th></th>
<th>Amblyopic eye 162.5 ± 178.7</th>
<th>Amblyopic eye 157.4 ± 171.6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-amblyopic eye 42.2 ± 46.2</td>
<td>Non-amblyopic eye 70.3 ± 77.0</td>
</tr>
</tbody>
</table>

Values expressed as mean ± standard deviation.
value corresponds to a light stimulus of greater intensity.) Threshold for the non-amblyopic eye was "statistically significantly" different from the normal control patients (p=0.002, two sample t-test). Threshold sensitivity in the amblyopic eyes was "very" (specifically defined as p=0.001 or less) "statistically significantly" different from the matched non-amblyopic eye (p=0.000, paired t-test) and from normal control threshold levels (p=.000, two sample t-test). However, a correlation between the threshold in the amblyopic eye and that in the non-amblyopic eye did not reach a "statistically significant" level (p=0.94, Pearson correlation). That is, eyes with reduced sensitivity in the amblyopic eye did not all have a commensurately reduced sensitivity in the non-amblyopic eye, with a probability better than 0.05.

In 25 of 26 anisometropically amblyopic patients, a scotoma was measured in the amblyopic eye. The mean scotoma area was 162.5 ±178.7 arc-min² with a range of 0.0-512.5 arc-min² (see prior Table II).

**Twenty of these 26 anisometropically amblyopic patients had a relative scotoma in the fellow, non-amblyopic eye.** The other 6 patients did not show a scotoma in the fellow eye. The mean scotoma size, including the six patients not showing a scotoma, was 42.2 ±46.2 arc-min² (range 0-150.2 arc-min²). Average scotoma size, excluding the six patients who did not have a scotoma, was 54.8 ±45.5 arc-min² (range 4.95-150.2 arc-min²). The SLO images of two patients with anisometropic amblyopia with scotoma areas delineated are shown in **Figures 4 & 5. on the following two pages.**

In this group, the area of the scotoma in the anisometropically amblyopic eye was, on average, 3.9 times larger than the scotoma, if any, in the fellow eye (p=.001, paired t-test, see **Figure 6, below**). Of only those patients with a

![Figure 6](image_url)
scotoma in the fellow eye (20/26), the scotoma in the amblyopic eye was, on average, 3.7 times larger than the scotoma in the fellow eye (p=.002). This includes five patients where the scotoma in the normal fellow eye measured LARGER than that in the amblyopic eye, but was mapped at a lower stimulus intensity than that necessary to evoke a response in the amblyopic eye (i.e., the stimulus to elicit a response in the presumed normal fellow eye was much dimmer than that necessary in the amblyopic eye). There was a correlation between the scotoma size in the amblyopic eye and that in its fellow eye (p=.048, Pearson correlation, just marginally "statistically significant"). That is, patients with larger sized scotomata in the amblyopic eye had, on average, a larger sized scotoma in the fellow eye.

One patient did not have an identifiable scotoma in either the amblyopic or the non-amblyopic fellow eye. There were no distinct clinical features of this patient that differed patently from the group as a whole. Visual acuity was 20/20 in the normal non-amblyopic eye and in the amblyopic eye visual acuity improved from 20/40 to 20/20 with treatment. Stereovisual acuity was 50 seconds of arc (i.e., "normal") at the end of treatment.

Data was therefore analyzed to determine if any features of the SLO scotoma findings correlated with amblyopia patient clinical treatment outcomes. There was a correlation between amblyopic eye scotoma area and the interocular visual acuity difference before treatment (p=.034) and also after treatment (p=.000, Pearson correlation). This correlation also existed in the non-amblyopic eye before (p=.048) and after treatment (p=.003, Pearson correlation). That is, scotomata areas in the amblyopic and non-amblyopic eyes were larger in patients with greater interocular visual acuity differences before treatment and after treatment.

There were "statistically significant" correlations between the scotoma size in the presumed normal non-amblyopic eye and the visual acuity of the amblyopic eye. Scotoma size in the presumed normal non-amblyopic eye was larger in those patients whose amblyopic eye visual acuity was poorer both before treatment (p=.035) and after treatment (p=.003, Pearson correlation).

As a group, patients with larger amblyopic eye scotomata had a poorer LogMAR amblyopic eye visual acuity at the start (p=.034) and at the end of amblyopia treatment (p=.001, Pearson correlation). Such a correlation did not exist in the normal non-amblyopic eye where scotoma size was not "statistically significantly" correlated with pre-treatment (p=.490) or post-treatment visual acuity (p=.285) of the amblyopic eye.

To further evaluate whether SLO findings correlated with amblyopic clinical features, two subgroups of patients were assessed. One subgroup comprised the 7 patients who had the best final visual acuity in the amblyopic eye and the other subgroup comprised the 7 patients who showed the poorest final amblyopic eye visual acuity. (Seven patients were chosen because 5 patients had the "third poorest" final visual acuity in the amblyopic eye.)

In the two subgroups of patients with anisometropic amblyopia, those who showed the poorest final visual acuity in the amblyopic eye (LogMAR 0.57 ±0.17 lines, n=7; Snellen equivalent 20/74) had "statistically significantly" larger macular scotomata in both the amblyopic and non-amblyopic eye than those found in the patients whose amblyopic eye final visual acuity was among the best (0.03
Relative Scotomata in the "Normal" Eye of Functionally Amblyopic Patients

D. A. Johnson, MD, PhD

SLO thresholds and clinical outcomes in the two "experimental" subgroups. Threshold in the amblyopic eye of the better outcome patients (17.4 ±2.8 dB) was not "statistically significantly" different from that found in patients with poorer amblyopic eye final visual acuity (17.0 ±5.5 dB, p=.859). Threshold was also not "statistically significantly" different between the presumed normal non-amblyopic eye of the better outcome group (22.0 ±1.8 dB) versus the poorer outcome patients (23.6 ±3.0 dB, p=.266).

Data was similarly analyzed for the subgroups of amblyopic patients who showed the greatest number of lines of visual acuity improvement in the amblyopic eye (6.85 ±1.42 LogMAR lines, n=5) versus the subgroup with the fewest lines of improvements (0.16 ±0.29 LogMAR lines, n=5). There did not appear to be any features of the SLO data that correlated with visual acuity outcome in terms of number of LogMAR lines improved. Between these two subgroups, there was no "statistically significant" difference in threshold in the amblyopic eye (p=.952) or non-amblyopic eye (p=.363). Nor was there any "statistically significant" difference between amblyopic patients who improved the greatest or fewest number of lines of visual acuity in terms of the scotoma size of the amblyopic eye (p=.847) or presumed normal non-amblyopic eye (p=.754).

As a group, scotoma area in the amblyopic or non-amblyopic eye was not correlated with level of stereoacuity. Patients who had high (i.e., normal) levels of stereopsis (a stereoacuity of 50 seconds of arc or better) were compared to those who showed no measurable stereopsis. Six patients had excellent stereopsis (a stereoacuity in 5 of them of 40 seconds of arc; the sixth, of 50 seconds of arc). Eight anisometropic patients had no measurable stereopsis. Scotomata in both the amblyopic and non-amblyopic eyes were larger in these 8 who had no stereopsis. The scotoma area in those with good stereopsis averaged only 37.4 ±33.4 arc-min² in the amblyopic eye and 10.9 ±12.1 arc-min² in the presumed normal non-amblyopic eye whereas the scotoma areas in the amblyopic and non-amblyopic eyes of patients with no stereopsis were much bigger in area, averaging 209 ±207 arc-min² (p=.055) and 58.2 ±60.5 arc-min² (p=.68), respectively. Remarkably, this huge difference was only marginally (p=0.055) "statistically significantly" different due to the large standard deviations noted.

Patient age was not a factor in this study in terms of either response to amblyopia treatment or performance on SLO testing. There was no correlation between age and pre- or post-treatment visual acuity, number of LogMAR lines improved or interocular visual acuity difference. This was true of both the amblyopic and the non-amblyopic eye. Similarly, there was no correlation between age and SLO results (threshold or scotoma area) in either the amblyopic or the non-amblyopic eye.

In summary, for these anisometropic amblyopia patients, there was:

1. an inverse correlation between the size of the amblyopic eye scotoma and the quality of amblyopic eye visual acuity both prior to initiation of amblyopia treatment and at the end of treatment.

2. a positive correlation between scotoma size and number of lines difference between the amblyopic eye and the presumed normal fellow eye both at the start and end of treatment. Patients with larger amblyopic eye scotomata areas had commensurately larger scotomata in the fellow, non-amblyopic eye. Those patients whose amblyopic eye had better final visual acuities had smaller macular scotomata in both the amblyopic and presumed normal non-amblyopic eyes than did those patients whose final amblyopic eye visual acuity was poorer.
Strabismic Amblyopia Patients

Twenty (9 male, 11 female) were treated clinically and evaluated with the SLO (see Table I, prior pages). The mean age of amblyopia diagnosis and treatment was 62.7 ±29.6 months (range 15.0-129.0 months). Followup averaged 49.4 ±25.1 months (range 12.0-91.0 months). Mean age at SLO testing was 101.3 ±19.3 months (range 71.0-142.0 months). The right eye was the amblyopic eye in 8 patients; the left eye in 12 patients.

Seven of these 20 patients had an accommodative esotropia type of strabismus, which was well controlled with spectacle correction and surgery was unnecessary. The other 13 of these 20 patients had either a non-accommodative deviation or a mixed accommodative and non-accommodative deviation for which surgery had been performed. In this latter group, only 1 patient was exotropic. The others were all esotropic. The preoperative deviation averaged 31.1 ±9.5 prism diopters (range 20.0-45.0 prism diopters). All patients in this group had a deviation of 10 prism diopters or less on alternate prism cover testing at final followup.

For this group, the pre-treatment visual acuity in the amblyopic eye was LogMAR 0.48 ±0.15 (Snellen equivalent approximately 20/60) with a range of LogMAR 0.18 - 0.70 (see Table II) and pre-treatment fellow eye visual acuity was LogMAR 0.10 ±0.08 (Snellen equivalent approximately 20/25), with a range of 0.0-0.3 LogMAR. After treatment, the visual acuity in the amblyopic eye had improved, on average, 2.35 ±1.8 LogMAR lines (range 0.00-6.00 lines; p=.000), yielding a post-treatment visual acuity of LogMAR 0.25 ±0.13 (range 0.10-0.48; approximate mean Snellen equivalent 20/36; see also Table II). Normal fellow eye acuity post-treatment was LogMAR 0.06 ±0.05 (range 0.0-0.1; Snellen equivalent 20/23), reflecting a change of 0.50 ±0.70 LogMAR lines (range 0.0-2.0 LogMAR lines; p=.009).

There was a "statistically significant" difference between the interocular difference in visual acuity before and after treatment. Prior to amblyopia therapy, the mean interocular difference was 3.81 ±1.47 LogMAR lines. After therapy, this difference was 1.97 ±1.23 LogMAR lines (p=.000, paired t-test), reflecting an average improvement of interocular visual acuity difference of 1.85 ±1.74 lines (range 0.0-6 lines; Table II).

Stereopsis was measurable in 10 patients, but all but two were poorer than 100" of arc. One achieved a normal 40" of arc stereopsis after strabismus surgery. The other had 100 secs of arc stereopsis.

Threshold sensitivity averaged 17.3 ±5.8 dB (range 0-24 dB) in the amblyopic eye and 21.6 ±3.3 dB (range 15-27 dB) in the non-amblyopic eye (Table II). Threshold sensitivity was reduced compared to control patients in both the amblyopic eye (p=.000, two sample t-test) and the non-amblyopic eye (p=.000, two sample t test). There was a "statistically significant" difference in threshold sensitivities between the amblyopic and non-amblyopic eyes of the same patient (p=.008, paired t-test, see Figure 7, below). Also there was a
also found to have a scotoma in the fellow eye, averaging 70.3 ±77.0 arc-min² (range 9.75-274.9 arc-min²). The SLO images of a patient with strabismic amblyopia are shown in Figure 9, next page.

For this group, the area of the scotoma in the amblyopic eye was 2.23 times larger than that in the fellow eye (p=.038, paired t-test; see Figure 10, below). However, in 3 patients, the scotoma in the normal fellow eye was actually

"statistically significant" correlation between the two eyes, in that a lower threshold level in the amblyopic eye correlated with a lower threshold level in the non-amblyopic eye (p=.022, Pearson correlation; see Figure 8, above).

In all 20 patients, a scotoma was found in the amblyopic eye, averaging 157.4 ±171.6 arc-min² with a range of 23.3-699.6 arc-min² (Table II). All 20 strabismic patients were

**Figure 8 (Johnson DA):** Strabismic amblyopia: Scatterplot of the amblyopic eye and non-amblyopic eye thresholds. A best fit regression line is shown (p=.022, Pearson correlation).

**Figure 10 (Johnson DA):** Boxplot of the SLO scotoma areas of the normal non-amblyopic eye (left) and amblyopic (right) eyes of strabismic amblyopia subject-patients. The shaded box represents the middle 50% of observations with the horizontal line in the shaded area indicating the median data value. The lines extending vertically from the box extend to indicate the range: the lowest and highest values of the data.
Figure 9 (Johnson DA): SLO images of a patient with strabismic amblyopia. After determining threshold, scotomata were delineated in the right, non-amblyopic eye (top) and left, amblyopic eye (bottom). This patient's visual acuity in the non-amblyopic eye was 20/25. The amblyopic eye visual acuity was 20/60 and did not improve with conventional amblyopia treatment. The patient demonstrated no stereopsis before or after amblyopia treatment.
larger than that in the amblyopic eye. But, in each case, the amblyopic eye required a greater stimulus intensity to evoke a response than did the normal eye. There was not a correlation in this group between scotoma size in the amblyopic and non-amblyopic eyes (p=.426, Pearson correlation).

Although there was not a "statistically significant" correlation between the visual acuity in each amblyopic eye at the start of amblyopia treatment and the acuity in that eye after treatment (p=.535, Pearson correlation), there was a correlation between the visual acuity of the amblyopic eye before treatment and the number of lines of acuity improved by therapy (p=.003, Pearson correlation). This is not surprising in that it might be expected that those eyes with poorer initial visual acuity will show a greater improvement, in terms of lines of acuity improved, than those eyes with better pre-treatment acuity. Also, there was a correlation between the number of LogMAR lines between the amblyopic and fellow eyes and the number of LogMAR lines improvement in the amblyopic eye with treatment (p=.006, Pearson correlation). That is, the greater the pre-treatment interocular acuity difference, the greater the improvement in LogMAR lines of visual acuity.

In the amblyopic eye, threshold level and scotoma size were not linearly related (p=.720, Pearson correlation). However, there was a correlation between threshold sensitivity and scotoma area in the non-amblyopic eye. Patients who required a more intense stimulus for threshold detection were found to have larger scotoma areas (p=.008).

Analysis was done to determine if SLO findings correlated with the clinical findings of patients with strabismic amblyopia. There was a "statistically significant" correlation between the size of the amblyopic eye scotoma and the LogMAR line difference between the eyes at the start of treatment (p=.002, Pearson correlation) but not after treatment (p=.129, Pearson correlation). There were no correlations between scotoma area in the non-amblyopic eye and any visual acuity features of the amblyopic eye, including amblyopic eye pre-treatment visual acuity (p=.809), post-treatment visual acuity (p=.660) or lines of acuity improved (p=.915, Pearson correlation).

The subgroup of the 5 patients with the best final visual acuity in the amblyopic eye (LogMAR 0.10 ±0.0, Snellen equivalent 20/25) was compared to the 5 patients whose amblyopic eye showed the poorest final visual acuity (LogMAR 0.41 ±0.07, Snellen equivalent 20/51). There was no "statistically significant" difference between the groups in threshold sensitivity of the amblyopic eye (p=.50, 2 sample t-test) or the non-amblyopic eye (p=.085, 2 sample t-test).

There was a greater difference in threshold sensitivity between the amblyopic and non-amblyopic eyes of patients with poorer final amblyopic eye acuity (p=.004, paired t-test) as opposed to that difference in patients with better final acuity (p=.178, paired t-test). That is, patients whose final visual acuity in the amblyopic eye was poorest after treatment had a greater difference in threshold sensitivity between the amblyopic and non-amblyopic eyes. No features of SLO scotoma size were found to correlate with final visual acuity outcome in either the amblyopic or non-amblyopic eye for either the best or worst final acuity subgroup. Nor were any of these SLO findings correlated with stereopsis.

When the patients were subdivided into those 5 patients whose amblyopic visual acuity increased the greatest number of lines (4.84 ±1.08 LogMAR lines) and those who had the fewest number of lines improvement (0.4 ±0.55 LogMAR lines), no correlations were found among SLO results.

Otherwise, there were no "statistically significant" correlations between the scotoma size in either the amblyopic eye or the fellow eye, in each of the following: amblyopic eye acuity at start or end of treatment, fellow eye acuity at start or end of treatment, treatment duration, number of LogMAR lines improved with treatment, difference in acuity between the eyes at the end of treatment, age at which treatment was begun or ended or age at which the SLO was performed.

Patient age did not appear to be an important factor in the response to amblyopia treatment. There was no correlation between age and pre- and post-treatment visual acuity, number of LogMAR lines...
acyuity improved or interocular visual acuity. Age also did not appear to be a factor in SLO performance. No correlation was found between patient age and threshold in the amblyopic (p=.532) or non-amblyopic (p=.088) eye. Nor was age correlated to scotoma area in the amblyopic (p=.291) or non-amblyopic (p=.605) eyes.

**Comparison of Anisometropic to Strabismic Amblyopia Groups**

The findings of those patients with anisometropic amblyopia were compared and contrasted to those of strabismic amblyopia patients. Although the anisometropic patients were, on average, slightly older than the strabismic patients when diagnosis was made and treatment begun (66.5 months and 62.7 months, respectively), there were no "statistically significant" differences between the two groups in terms of age at start of treatment (p=.63, two sample t-test) or age at which treatment ended (p=.72, two sample t-test, Table II). The strabismic group had a longer average followup than the anisometropic group (49.4 months and 47.8 months, respectively), but this also was not "statistically significantly" different (p=.84, two sample test). Treatment duration was not "statistically significantly" different between the two groups (p=.98). Nor was there a "statistically significant" difference between the two groups in terms of age at which the SLO was performed (p=.64, two sample test).

More patients in the anisometropic group had some degree of stereopsis (69.2%) than in the strabismic group (50%). However, only three strabismic patients reached a stereocuity of 100 seconds of arc or better, while 6 of the anisometropic patients achieved 50 seconds of arc or better, 5 achieving a normal 40 seconds of arc.

There was no "statistically significant" difference between the groups in terms of the non-amblyopic eye visual acuity at the start of treatment (p=.95) and number of lines improved in the non-amblyopic eye (p=.28, two sample t-test). However, because the anisometropic group showed slightly greater improve-ment from treatment start to final followup, the difference between the groups reached "statistical significance" in terms of final visual acuity in the non-amblyopic eye (p=.006). Two qualifications are important. First, the actual clinical difference in final visual acuities is quite small (anisometropic, LogMAR 0.015; strabismic, LogMAR 0.055; Snellen equivalents, 20/21 vs. 20/23 respectively). Second, in some patients, the initial pretreatment acuity was recorded using a projected pictures test. Not only might this acuity testing method be less precise than letter recognition, these patients were considerably younger at the entry point. Thus, the apparent "statistically significant" difference most likely has little or no clinical relevance or im-portance.

In terms of the visual acuity in the amblyopic eye, there was a "statistically significant" difference between the two groups at the start of treatment (p=.009), but not at final followup (p=.314, two sample t-test), although the mean acuity of the amblyopic eyes in the aniso-metric group was poorer than the strabismic group at each time point in the study.

There was a "statistically significant" difference between the two groups in terms of the number of LogMAR lines difference between the amblyopic and fellow eyes at the start of treatment (p=.005), and also at the end of followup (p=.095), with the anisometropic patients having a greater pretreatment LogMAR line visual acuity difference between the eyes (5.53 ±2.41 LogMAR lines vs. 3.81 ±1.47 LogMAR lines, respectively). On average, the amblyopic eyes in the anisometropic group showed a greater LogMAR line improvement than those in the strabismic group (3.51 lines vs. 2.35 lines, p=.06, two sample t-test).

Thus, although patients with anisometropic amblyopia had a poorer visual acuity level in the amblyopic eye pre-treatment and a greater interocular visual acuity difference between the amblyopic and the non-amblyopic eye, these eyes showed a significantly greater improvement with treatment such that amblyopic eye visual acuities of an-isometropic and strabismic patients were not "statistically significantly" different post-treatment.

There was no "statistically significant" difference between the two groups for SLO threshold in the amblyopic eye (p=0.74) or the fellow, non-amblyopic eye (p=0.31). It is of note that in both aniso-metric and strabismic amblyopia pa-tients, threshold
stimulus intensity for recognition in both the amblyopic and non-amblyopic eyes was at greater light intensities than in the normal control patients.

There was no "statistically significant" difference between mean scotoma area in the non-amblyopic eye in anisometric patients compared to strabismic patients (42.2 vs. 70.3 arc-min², p=0.16, two sample t-test), although the normal fellow eye scotoma in strabismic patients tended to be larger. Nor was there a "statistically significant" difference between amblyopic eye scotoma areas in anisometric patients (mean 162.5 arc-min²) versus strabismic patients (mean 157.4 arc-min²; p=.92). There was a "statistically significant" difference between the two groups in terms of scotoma size related to stereopsis. When anisometric patients were sub-divided based on stereopsis, those patients with better stereopsis had smaller macular scotomata in both the amblyopic and non-amblyopic eye than did those with no stereopsis. No such correlation existed among strabismic amblyopia patients.

In summary, among all features analyzed, the two experimental groups were "statistically significantly" different in only several areas: stereopsis (better in the anisometric group), visual acuity in the amblyopic eye pre-treatment (poorer in the anisometric group), post-treatment visual acuity in the non-amblyopic eye, and LogMAR lines different at start (greater difference in the anisometric group) but not at final followup.

END of RESULTS SECTION

SUMMARY-DISCUSSION

In this study, a selected collection of 46 patients that had been treated for anisometric or strabismic amblyopia was evaluated with the Scanning Laser Ophthalmoscope after reaching optimal visual acuity in the amblyopic eye. Forty-five of the 46 patients who were tested with the SLO showed a macular scotoma in the amblyopic eye. Forty of the 46 were also found to have a relative scotoma in the contralateral fellow, presumed "normal" eye. In contrast, no scotoma was identified in either eye of any of the 10 normal control patients.

Scanning Laser Ophthalmoscopy (SLO)

Developed in the late 1970s-1980s, SLO has shown increased clinical utility in the last 10-15 years. Its use has been shown to be of benefit in optic nerve analysis (12,13), retinal assessment of macular lesions (14,15), assessment of retinal foci that underlie metamorphopsia (16-18), and microperimetry (19-21), as was its use in this study.

The SLO uses a weak laser beam to scan the retina, generating a raster (scanning) image of the retina that can be viewed on a monitor. Simultaneously, a second HeNe laser (545 nM) can project a stimulus of variable size, intensity and duration directly onto the retina. It is directed by the examiner, guided by a real-time view of the retina, including reference points set to control for eye or patient movement (23). Pupillary dilatation is not required for macular viewing on the SLO.

As a microperimetry tool, the SLO has been most extensively used in low vision evaluation of patients with macular lesions, especially age-related macular degeneration (19-21). Affected patients may develop foveal scotomata, resulting in the development of an extra-foveal preferred retinal locus (PRL) of fixation. The SLO allows the examiner to determine that portion of the retina the patient is using for fixation. If it is found that another retinal area provides greater sensitivity, the patient may be "trained" to re-fixate with this more sensitive retinal locus.

In this study, the SLO was used to assess for the presence, size and depth of scotomata in patients with amblyopia of various types and contrast these findings with a control group. This data, along with data acquired by typical ophthalmologic examination assessment, was assessed overall and within groups based on amblyopia etiology, to evaluate whether any factors were associated with either amblyopia type or treatment outcome.

SLO Evaluation of Amblyopia

Amblyopia was defined using generally accepted current criteria; that is, in preverbal children, a strong fixation preference for one eye over the other, or, in verbal children, the demonstration, using age-appropriate testing methods, a two line
or greater visual acuity difference between the eyes. Patients were therapeutically addressed utilizing standard conventional current treatment modalities, including occlusion and/or atropine penalization. Eye muscle surgery was performed in those patients with a non-accommodative strabismic deviation. Appropriate spectacle correction was instituted in patients with anisometropia or accommodative esotropia.

**Regarding All Amblyopia Patients:**

Forty of the 46 patients evaluated showed an improvement in visual acuity in the amblyopic eye in response to currently conventional amblyopia treatment. In the other 6 patients (13%), the visual acuity did not change from entry level under treatment. No amblyopic eyes showed a decrease in visual acuity during the treatment period.

As might be expected, a scotomatous defect was found in the amblyopic eye of almost all patients. Only one anisotropic patient did not have a detectable scotoma in the amblyopic eye. While true physiologic mechanisms may underlie the variation in scotoma sizes, several factors are believed to account for the wide range of scotoma dimensional values obtained. Primary among these is the fact that this is a psychophysical test being performed in children. While our subject-children were selected based on their perceived ability to maintain steady fixation at the scanning laser ophthalmoscope, any such test in children is subject to their ability to follow instructions, to focus mentally and concentrate, and maintain visual fixation on the target. Overall, the patients were felt to have performed the SLO very well. Mapping of the scotoma on the SLO also lacks some precision similar to that of Goldmann kinetic perimetry, in that this is a "best-fit" approach based upon patient response and the SLO operator-examiner's interactive mapping of responses.

However, compared to other similar psychophysical tests, SLO microperimetry is extremely precise in terms of stimulus delivery location and centration of data on the final image. This accrues from the macula being viewed in real time while testing, so that a stimulus is not delivered unless the patient is seen to be fixating properly in real time. Thus, there is no measure of perimetric "fixation loss" because a stimulus is not delivered if visual fixation is not central.

This study’s **major new finding** is that a heretofore, completely unknown and even unhypothesized, relative scotoma was discovered to be present in the fellow and presumed totally "normal" contralateral eye of 40 of 46 (87% of) treated functionally amblyopic patients.

In the group as a whole, the scotoma size was, on average, more than 2.5 times greater in area in the amblyopic eye than in the normal fellow eye. In 8 of these patients the normal fellow eye scotoma measured a larger area than that in the amblyopic eye. However, in these eight cases and in all cases, the threshold for the testing stimulus in the amblyopic eye was higher, i.e., of greater intensity than that required in the fellow normal eye. While this confounds the data somewhat, it was necessary to increase the stimulus threshold intensity for accurate measurement; this would only underestimate the mean difference between the two eyes.

**Anisometropic Amblyopia Patients**

All but 3 of the 26 patients with anisometropic amblyopia had improvement in visual acuity in the amblyopic eye after treatment, averaging an increase of 3.51 LogMAR lines improvement. Although there may have been small variations in amblyopia treatment strategies employed among this group of patients, the level of improvement compares favorably to other prior studies of amblyopia treatment (24, 25). In the 2002 Pediatric Eye Disease Investigator Group (PEDIG) study comparing atropine penalization to occlusion of the normal fellow eye (25), visual acuity improved 3.16 LogMAR lines in the occlusion group and 2.84 LogMAR lines in the atropine group. These results are comparable to the overall improvement of 3.51 LogMAR lines in this study. It should be noted that our current study had a broader initial amblyopic eye visual acuity inclusion criteria than the PEDIG study which limited inclusion to amblyopic eye visual acuity to no poorer than 20/100. Fifteen of the 26 anisometropic patients in this study achieved a final Snellen visual acuity of 20/40 or better in the amblyopic eye; only 5 of the 15, however, (19% of the
whole group) could read to the Snellen 20/20 level. One of these five 20/20 patients had no scotoma in either the amblyopic or non-amblyopic eye. Three of these five 20/20 patients had no scotoma in the normal non-amblyopic fellow eye and their amblyopic eye scotomata were "statistically significantly" smaller than those of the anisometropic group as a whole. These five 20/20 patients were among the six anisometropic patients with the best final stereopsis (40 secs of arc in 5 patients, 50 secs of arc in the other). Assessing all other measured clinical features of these 5 patients failed to reveal any "statistically significant" differences from the anisometropic amblyopia group.

While all but one of the 26 patients with anisometropic amblyopia had a scotomatous defect in the amblyopic eye, 20 of the 26 were found to have a scotoma in the fellow, presumably normal, eye, despite an average normal visual acuity in the non-amblyopic eye of LogMAR 0.015 (Snellen equivalent 20/20.7). This included 3 patients who had stereopsis of 40 seconds of arc or better.

The amblyopic eye scotoma was, on average, almost four times greater in area than that in the fellow presumed normal eye. There was a direct correlation between the scotoma size of the amblyopic eye and that of the fellow eye; a larger scotoma in the amblyopic eye was predictive of a larger scotoma in the normal fellow eye. Scotomata areas in the amblyopic and non-amblyopic eyes were also larger in patients who had greater interocular visual acuity differences before and after treatment. Further, anisometropic patients who had poorer visual acuity outcomes in the amblyopic eye had larger amblyopic and non-amblyopic eye scotomata than those patients who had better visual acuity outcomes. There did not appear to be any correlation between scotoma size (either in the amblyopic or fellow eye) and age at treatment, number of LogMAR lines difference in visual acuity at the end of treatment, number of LogMAR lines improvement in visual acuity, age at entry or age at which the SLO was performed, or treatment duration.

**Comparison of Anisometropic to Strabismic Amblyopia Patients**

One question of interest in this study was whether amblyopia treatment and outcome features or SLO characteristics were different between patients with anisometropic amblyopia as opposed to those with strabismic amblyopia.

There were no "statistically significant" demographic differences between the two groups. Pre- and post-treatment visual acuities also were not "statistically significantly" different between the groups, in either the amblyopic eye or the normal fellow eye.

More anisometropic patients had measurable stereopsis at final followup and
the quality of stereopsis was superior to the strabismic patients. Only 3 strabismic patients had stereopsis that reached 100 seconds of arc, while 12/26 anisometric patients had stereopsis of 100 seconds of arc or better. Five anisometric amblyopia patients had 40 seconds of arc and better stereopsis correlated with smaller scotoma in the amblyopic and non-amblyopic eyes. This difference may stem from one of the fundamental differences in the underlying cause of the amblyopia. In those patients with anisometropia, the image, although degraded in the amblyopic eye, remains in binocular alignment with, and therefore potentially fusible with, the fellow eye, whereas in the strabismic patients, the visual axes are misaligned and, regardless of binocular surgical alignment, establishment of fusion and stereopsis may be reduced or impossible.

There was a greater pre-treatment interocular visual acuity difference in the anisometric patients than in the strabismic patients, and the former group did not show a "statistically significantly" greater LogMAR line improvement with treatment than the latter group. However, the finding that the anisometropic patients had better stereoaucity than the strabismic patients further supports the idea that potentially fusible, binocularly aligned eyes, despite relatively greater degraded image clarity in the amblyopic eye, have the higher potential for superior stereopsis, as was seen in the anisometric patients.

Threshold levels for stimulus recognition were almost identical in the amblyopic and non-amblyopic eyes of the 2 groups. In terms of scotoma size measured in either the amblyopic eye or the fellow eye, there were no "statistically significantly" differences between the groups. However, the interocular difference between amblyopic and fellow eye scotoma size was greater in the anisometric patients than in the strabismic patients. One might postulate that this may stem from a greater interocular visual acuity difference in the anisometric patients and this was, in fact, the case, both prior to treatment and at final follow-up. Thus, although the potential for better stereopsis may be greater in patients with a binocularly fusible, although degraded, image in the poorer seeing eye, the data suggest that such a situation may not result from, or result in, a smaller interocular scotoma area difference.

**Scotoma Features in the Amblyopic and Non-Amblyopic Eyes**

A relative scotoma in an amblyopic eye is not unexpected. Electrophysiological examination of amblyopic patients shows findings indicative of a scotoma in the amblyopic eye. Fioretto & colleagues (26) evaluated 8 patients with dense amblyopic in one eye using pattern electroretinogram (PERG), pattern visual evoked potential (VEP) and event-related potentials (ERP), in association with computerized perimetry, to assess scotoma presence and features. PERG traces from the amblyopic eyes were irregular in morphology and of reduced amplitude relative to both the non-amblyopic eye and normal control patients. This finding suggests some inherent defect in amblyopic eyes at the level of the retina, specifically, the ganglion cell and amacrine cell layers. However, their study also showed similar response profiles when the non-amblyopic eye was blurred with filters to the acuity of the amblyopic eye. Thus, they concluded, if there is a functional defect at the level of the retina, it is likely minor in that simple form degradation produced similar results.

VEP assessment of the amblyopic eyes found significant reductions in amplitude and latency relative to the controls. Such a test cannot differentiate a true conduction pathway deficit (which seems unlikely) from a more significant deficit at the level of the lateral geniculate nucleus (LGN), as has been previously demonstrated to be one of the underlying abnormalities in amblyopia. The abnormal ERP findings suggest a higher level component to the deficit in amblyopia. Thus, the fundamental defect in amblyopia may be multifactorial, but predominantly localizable to abnormal binocular interactions at the level of the lateral geniculate and ultimately reflected by abnormal binocular interactions at the level of the visual cortex. As a result, one might predict a scotomatous defect on psychophysical testing. Such was the case in this study, as a scotoma could be mapped in the amblyopic eye of 45 of the 46 patients.

The threshold for stimulus recognition was higher in the amblyopic than the non-amblyopic eye in this study. Similar
findings were noted by Fioretto & colleagues (26) using automated perimetry. They found reduced sensitivity particularly in the central field. They attributed some of this to poor fixation. It should be noted that the patients in the Fioretto study, although older (aged 11-46 years) had denser amblyopia (approximately 20/100-20/250) than most of the patients in this study.

What was not anticipated on the basis of prior studies and knowledge was the presence in these amblyopic patients of a scotoma in the fellow, presumably "normal" eye. This was present in 40 of the 46 patients in this study. In the 6 patients who did not demonstrate a normal non-amblyopic eye scotoma, there were no apparent clinical features which distinguished them from other patients in the cohort other than that they had among the better stereocautities and smaller amblyopic eye scotomata of patients tested. However, other patients, who did have non-amblyopic eye scotomata showed similar clinical characteristics.

One might also expect there to be some correlation between amblyopic eye scotoma size and visual function and/or interocular interactions. For anisometropic patients, scotoma sizes were larger in the amblyopic eyes with poorer acuity and when there was a larger interocular visual acuity difference. Both of these correlations held at pre-treatment and final followup. Such a correlation did not exist in the non-amblyopic eye, perhaps because of the relatively small range of pre- and post-treatment visual acuities. In strabismic amblyopia patients, there were no correlations, in the amblyopic or non-amblyopic eye, between scotoma size and pre- or post-treatment visual acuity or number of lines of improvement. There was a direct correlation between scotoma areas in the amblyopic and the non-amblyopic eyes of anisometropic patients but not among strabismic amblyopia patients. Stereopsis, as an indicator of interocular interaction, was significantly different between the two groups with better stereopsis among anisometropic patients. Further, anisometropic patients with better stereopsis had smaller amblyopic and non-amblyopic eye scotomata, suggesting that scotoma size was smaller when binocular interactions were greater. Thus, despite form degrad-
previously affected eye and restore a more normal anatomical and physiological character to the ocular dominance column organization (31,32).

Interestingly, damage to one eye after the critical period results in loss of cortical metabolic activity in columns driven by that eye (as would be expected), but no shrinkage in the corresponding ocular dominance column. Horton & co-workers (33) and Hendrickson & colleagues (34), using cytochrome oxidase (CO) histochemical techniques as a marker of patterns of metabolic activity, found that, in normal monkeys, levels of CO activity are homogeneous throughout layer IVc in the striate cortex, corresponding to equal ocular input to each column. However, after removal of one eye, they found a pattern of alternating dark and pale similarly sized columns, representing the normal and the affected eye.

Subsequently, Horton and Hedley-Whyte (35) noted similar findings in post-mortem examinations of humans. In patients with a history of a loss of one eye, even if that loss occurred as much as 20 years or more before death, but after the critical period of early visual development, there was a mosaic of alternating dark and light CO-labeled columns. In 1993, Horton & Stryker (11) reported another case evaluating ocular dominance column appearance in humans. A 53 year old male, with documented anisometropic amblyopia first diagnosed at approximately 5 years of age had an amblyopic eye visual acuity of 20/400 (refractive difference between the eyes was approximately 6 diopters). He subsequently became blind in his non-amblyopic eye from a metastatic lesion compressing the optic nerve. Post-mortem histochemical analysis showed that the ocular dominance columns of the two eyes were essentially the same size. Cytochrome oxidase staining (indicative of metabolic activity) was absent in the columns corresponding to the previously normal, but subsequently blinded eye, and present but reduced in width and not occupying the full width of the column in the amblyopic eye. These findings suggest that, once established, ocular dominance columns may persist anatomically, if not functionally, even in the absence of retinal input.

Horton, Hocking and Kiorpes (33) studied a single monkey with naturally occurring anisometropia and a behaviorally assessed visual acuity approximately 20/60 in the amblyopic eye. Using autoradiographic techniques, there was preservation of clusters of cortical cells arranged in columns corresponding to each eye. Although these columns were not shrunken, assessment of the same columns for cytochrome oxidase (CO) activity (and, hence, commensurate physiologic activity), revealed a pattern of dark central bands of positive stain separated by a pale gap of cells. Superimposition of the CO images onto the autoradiographs showed the dark bands to occupy the center of the ocular dominance columns and the paler bands to represent a transitional zone, which most likely represents the electrophysiologically less active binocular cell zones.

Kiorpes & associates (36) measured cortical neuronal responses in monkeys that had been experimentally rendered either strabismic or anisometropic. In all animals, cortical binocularity was reduced. As had previously been shown by Wiesel & colleagues (28), as well as others (30), columnar clusters of cells were identified that were strongly dominated by one or the other eye. However, they also encountered transition zones between the columns where neuronal units were difficult to drive visually, suggesting that these cellular regions represent binocular neuronal units, which, in normal monkeys contain the most strongly driven binocular cells, but, in their study, were absent.

While binocularly driven units were almost absent in the strabismic monkeys, the authors also found significantly few binocularly driven cortical neurons in the anisometropic animals. Quantitatively, there was not a “statistically significant” difference in proportion of binocularly driven neuronal units between the anisometropic and strabismic animals. However, differences in cortical neuronal unit response tended to follow a continuum related to severity of behaviorally measured depth of amblyopia. Although the reduction of cortical binocularly excited units was slightly greater in the strabismic animals, the overall physiological changes in cellular response were more directly correlated to the severity of the amblyopia rather than the etiology of the visual loss.
Although some of the above results may suggest that there had been shrinkage in the size of the ocular dominance columns in response to the experimental alterations, a number of studies indicate that some forms of amblyopia might not result in shrinkage of the ocular dominance columns (11,33).

These results are not in fundamental disagreement with the pioneering studies of Hubel & Wiesel and others that found shrinkage of ocular dominance columns in experimental amblyopia. In those early experiments, visual deprivation was total, achieved by suturing one eyelid closed or enucleation. Hence, there is a significant difference between total deprivational visual deficits and amblyopia secondary to either form degradation or spatial dislocation.

Other approaches that have been used to assess functional abnormalities in amblyopia include high resolution magnetic resonance imaging (MRI), blood-oxygen-level-dependent functional magnetic resonance imaging (BOLD fMRI), positron emission tomography (PET), and brain single positron emission tomography (SPECT). Kabasakal & co-workers (37) tested ten patients, aged 8-14 years, with known amblyopia (not specified as either strabismic or anisometric) using SPECT scans. While visually stimulating one or the other eye, patients were injected with a Technitium-labeled marker and imaged 20-30 minutes later. In all cases, they found greater occipital cortex activity accumulation when the normal eye was stimulated, as compared to the amblyopic eye. Demer & co-workers (6), using PET imaging, found that cortical blood flow and glucose metabolism were reduced in the primary visual cortex when monocular stimulation was applied to the amblyopic eye relative to stimulation of the non-amblyopic eye in strabismic amblyopia. Comparing the above two studies, PET scanning has better spatial resolution than SPECT scanning, although the clinical utility may not be practical or cost-effective in either case.

Evaluating normal and amblyopic eye responses to different contrast sensitivity stimuli, Goodyear & colleagues (38), using BOLD fMRI, found a consistently reduced level of stimulus-evoked response in areas of visual cortex when the amblyopic eye was stimulated as opposed to the response to the fellow eye stimulation.

In an effort to improve the resolution of neuroimaging methods, Goodyear & co-workers (39) used high-resolution fMRI to evaluate visual cortex response in human amblyopia. They found that, in adult patients with a history of early onset amblyopia, greater responses were found in areas corresponding to the unaffected eye versus the amblyopic eye. On the contrary, in two adult patients with later onset amblyopia, a shift in ocular dominance columns was not noted, further supporting the studies above, which suggest that, outside of the critical period of early visual development, ocular dominance columns are relatively static anatomically, despite reduced response to amblyopia eye stimulation. Choi & co-workers (5) found similar results of reduced activity in response to amblyopic eye stimulation, using fMRI in amblyopic patients ranging from 5-23 years of age.

**DIFFERENCES Between Anisometric and Strabismic Amblyopia VISUAL PATHWAYS**

A number of studies have found features of visual system structure and function that differ based upon the etiology of the amblyopia (11,33,36). It follows that because anisometropia generates an interocular discrepancy in form vision and strabismus generates a discrepancy in spatial localization, different processing pathways may be affected or preserved. Further, studies that have induced visual loss by deprivation (e.g., lid closure, corneal scarring (21,32)) may identify totally different alterations in visual function.

In this study, there were some contrasts between anisometric amblyopia and strabismic amblyopia such as stereopsis, pre-treatment visual acuity in the amblyopic eye, final visual acuity in the non-amblyopic eye and interocular LogMAR line visual acuity difference at the start, but not the end of treatment. However, for the most part, there were no “statistically significant” differences between the groups, especially in terms of presence and size of...
found some abnormalities in the fellow eye of amblyopic patients.

Furthermore, in some patients, these scotomata were present in one or both eyes AFTER what appears in all standard regards to have been 100% successful treatment of the amblyopia including normal visual acuity in both eyes and normal binocular cooperation as evidenced by a normal stereoaucy of 40 seconds of arc. So we may think we are in these best cases fully “curing” these children of their affliction. But the SLO tells us that that in most patients that is not true. Yes, in some cases which were clinically “cured” there were no scotomata in the normal non amblyopic eye after successful completion of treatment. But for the majority, it appears rather that there residual abnormalities in the upper visual pathways, which limit visual function probably permanently, albeit so mildly that the impediment is undetectable by conventional clinical testing of standard visual parameters.

Paralleling, perhaps predicting, the SLO findings in this study, Rogers & colleagues (42,43), while measuring contrast sensitivity in amblyopic children, found that the contrast sensitivity function (CSF) was reduced in the amblyopic eye even though that eye had achieved, with treatment, a visual acuity of 20/20. Their findings of diminished CSF in the fellow presumed "normal" eye of amblyopic patients after successful treatment of a contralateral amblyopia were not seen in their normal control patients. The decreased CSF was not induced by amblyopia treatment with occlusion. In fact, as the amblyopic eye visual acuity improved with treatment, the CSF in the non-amblyopic eye improved also. Thus, in both untreated and "successfully" treated unilateral functional amblyopia, the non-amblyopic eye is still not entirely normal.

In a followup study investigating this finding, Leguire and colleagues (43) found that there was a correlation between CSF frequency response and binocularity. They found greater (better) binocularity when there were smaller differences in CSF between the eyes and lesser (poorer) binocularity when interocular CSF differences were greater. Thus, these factors may all inter-relate in terms of necessary criteria.
for binocularity such that normal binocular alignment promotes lesser interocular CSF discrepancy and, hence, greater binocular interaction. This is supported by the greater degrees of binocularity noted in the anisometric patients in this, our study, also. Because so many visual cortical neuronal units are driven binocularly, it is reasonable to assume that the non-amblyopic eye might be affected by the amblyopic eye, as was proposed by Leguire (43). Disruption of binocularity by abnormal input from one eye would alter the function of binocularly driven cortical cells and thereby alter the measurable function of the fellow eye.

Other studies have also found abnormalities in the "normal" fellow eye of unilaterally amblyopic patients. See Rentschler & Hilz (44) who reported orientation sensitivity was abnormal in not only the amblyopic eye, but also in the non-amblyopic presumed normal fellow eye of strabismic patients.

The current study provides further evidence that the "normal" eye may not be truly normal in amblyopia. Leguire & colleagues (43) also followed CSF sensitivity before, during and upon completion of occlusion therapy.

This current study assessed all patients retrospectively and by SLO only at most recent followup after completion of amblyopia treatment. Thus, it is not known from our study whether there were changes in scotoma size in either eye during either amblyopia treatment. This then would indeed be the next research step in the investigation of SLO scotomata in amblyopia.

Other Study Limitations

While helpful in exploring the nature of amblyopia of different etiologies, some of the clinical approaches mentioned above, including that of this study using the SLO, suffer from technical limitations, particularly in data precision and resolution. Foremost among these is the age range of the patients of greatest interest. While some aspects of visual function in amblyopia can be assessed in more mature children and adults, important information would be gained by using "adult" techniques to analyze visual functional abnormalities of the early, developing visual system. Also, some of the tests were psychophysical in nature and, thus, will have some limitation of accuracy, especially in children. Psychophysical studies do not lend themselves to precise localization of the defect responsible for features of amblyopia in that the stimulus response represents the summed responses of the entire visual system, including higher order cognitive functions.

There are other limitations in this study, especially in the use of the SLO to evaluate features of amblyopia. The current study was retrospective, with data analysis at the end of followup after amblyopia treatment was felt to be clinically stable. As a result, while entry criteria were relatively uniform, amblyopia treatment approaches and specifics varied considerably among patients, as it does in any normal clinical practice, being readily adapted to each individual child and/or family. Furthermore, as in any study of amblyopia, since treatment is carried out primarily by the parents and at home, treatment compliance cannot be really accurately assessed. While each child in this group of patients was felt to have good treatment compliance, this could not be quantified. Indicative of less than perfect compliance is the fact that a number of patients had a change in treatment modality (occlusion to atropine or vice versa), and this was usually based upon issues of comfort and compliance.

The depth of the amblyopia was, in some patients, an underestimate. That is, there were four patients in whom the initial diagnosis of amblyopia was made while the child was still preverbal. Treatment was initiated and numerical visual acuity data only registered when the child became verbally cooperative later. Hence, a small number of patients may have had initial amblyopic eye visual acuity poorer than what was ultimately quantitatively tested and recorded.

Another limitation is that the patient selection process was not random. While there were certain criteria for inclusion in the data analysis, patients were chosen to perform the SLO only if the child’s performance at the slit lamp biomicroscope suggested that the child was sufficiently cooperative to perform the SLO test. This obviously eliminated younger patients.
Furthermore, further selection bias could result if there were a correlation between a child’s performance in our clinical setting and overall child and family compliance with treatment recommendations.

However, many aspects of the SLO testing, by virtue of the testing procedure itself, are very precise. Because microperimetry is performed while monitoring a live image of the macula, stimulus location was extremely accurate. Also, rather than relying on a computerized paradigm to assess fixation reliability, as is done in automated perimetry, the real-time monitoring of the macula assured that stimuli were not delivered unless fixation was on target.

Finally, the SLO data collection is similar to that obtained with the Goldmann perimeter. That is, the examiner performing the testing must identify levels of sensitivity and actively engineer a map of the scotomatous area based upon a finite number of testing points. The legitimacy of the data generated in this study was facilitated by the fact that the SLO was performed by an individual skilled in the testing and masked to all clinical information about the patients except name and age. By far, the most accurate data were generated by the computer-assisted measurement of the scotoma area, once it was plotted by the perimetrists. Such great precisional computational capability far exceeded the precision of the human scotoma mapping procedure.

REFERENCES
28. Wiesel TN, Hubel DH, Lam DNK. Autoradiographic


ABSTRACT: **Purpose:** To investigate the long-term vision outcomes of amblyopia treatment in “successfully”, compared with “unsuccessfully”, treated patients.

**Methods:** Forty-two participants (n=42, mean age 14.8 years, range 10-25 years) were enrolled in the study. Individuals with strabismic or mixed (strabismic and anisometropic) amblyopia were examined at a mean of 6.6 years (range 1-18 years) after cessation of amblyopia treatment. Participants were classified as being “successfully” treated (Group 1) if visual acuity of 6/7.5 or better was achieved at cessation of treatment, or “unsuccessfully” treated (Group 2) if visual acuity of 6/9 or less was achieved at cessation of treatment. Visual acuity was analyzed by calculating an interocular score or difference in visual acuity between the amblyopic and non amblyopic normal (control) eye.

**Results:** A deterioration of visual acuity occurred in 62% of the participants in both Groups 1 and 2. The mean deterioration of visual acuity over time for either group was less than one LogMAR chart line and was not "statistically significant" by convention (F [1,39] = 3.361, p=0.074). The outcomes achieved at cessation of treatment did not "statistically significantly" affect the mean deterioration that occurred over time (F [1,49] = 0.031, p=0.860).

**Conclusion:** Visual acuity was relatively stable over a mean followup period of 6.6 years. The treatment outcome and the success of amblyopia treatment were found to be irrelevant to long term stability of visual acuity. These findings suggest that amblyopia treatment mostly results in a lasting improvement in visual acuity, and that both unsuccessfully and successfully treated individuals maintain their visual acuity improvement achieved during treatment.
INTRODUCTION

Amblyopia affects between 1-4% of the population and is the most common form of vision impairment in children (1,2). In 1997, the effectiveness of amblyopia treatment in Great Britain was questioned by a report published by the National Health Service Center for Reviews and Dissemination (UK) (3). Specifically, and in fact, this report revealed primarily a lack of quality studies on the long-term effectiveness of amblyopia treatment. On the basis of this and other findings, the authors concluded that pre-school vision screening should be ceased.

Reports of deterioration in vision of successfully treated amblyopes vary widely in the literature. Studies indicate that between 17% and 76% of successfully treated amblyopes show regression in visual acuity at long-term followup (4-12). One of the earliest studies (5) reported that the majority of amblyopes lost approximately half of the vision improvement achieved during treatment, while 17% regressed to original pre-treatment levels. More recent studies have reported less deterioration, but variability amongst the results continues to exist. For instance, while Öhllson et al (10) found that visual acuity regressed in only 17% of amblyopic eyes at an average followup period of 10.4 years, Rutstein & Corliss (13) and the Pediatric Eye Disease Investigator Group (PEDIG) (14) recently reported somewhat higher rates. The PEDIG found that 24% of strabismic and anisometric amblyopes with a 1 year followup regress, while Rutstein & Corliss found regression in 35% of a similar amblyopic population with a mean followup of 8.2 years. The differences in the literature are possibly due to factors such as the inclusion or exclusion of amblyopes whose monitoring ended prior to 9 years of age, different followup periods, inconsistent definitions of amblyopia and a lack of controls for visual acuity measurements.

There has also been a lack of controls for the variables: (the) effects of practice and maturation. The need to control for maturation effects in longitudinal studies was highlighted by Levertovsky et al (7) who reported that while visual acuity deteriorated in 53% of amblyopic eyes, it also actually improved in 36% of amblyopic eyes after occlusion treatment had ceased. They suggested that this was due to the older age of the patients and their improved cooperation with the visual acuity test at long-term followup. In order to reduce these effects of maturation associated with longitudinal studies, visual acuity ratios that incorporate the non-amblyopic eye as a control for this factor should be used (4). However, only one study (4) has utilized this concept of interocular difference when investigating the regression of visual acuity in amblyopic patients. All other studies to date have analyzed visual outcomes for the amblyopic and non-amblyopic eyes separately.

To date, studies have also neglected to investigate the long-term stability of visual acuity in unsuccessfully treated patients. Studies on the natural history of untreated patients with amblyopia have reported that deterioration of visual acuity does occur over time (15,16). On this basis, Öhllson and colleagues (10) suggest that theoretically successful treatment of amblyopia could result in a "normalization" of the development of the amblyopic eye. If this is the case, unsuccessfully treated patients may show greater regression of vision than successfully treated amblyopes.

The aim of this study was to investigate the long-term vision outcomes of occlusion treatment in patients with successfully and unsuccessfully treated amblyopia. To do this, we compared the mean interocular visual acuity score achieved at cessation of treatment with that obtained at a long-term followup visit.

SUBJECTS and METHODS

Forty-two (n=42) individuals were included in this study. Participants were divided into two groups, successfully treated and unsuccessfully treated amblyopia. Group 1 comprised the successfully treated amblyopes. This outcome ("success") was defined as a visual acuity of 6/7.5 (20/25) or better at the cessation of treatment. Group 2 comprised the unsuccessfully treated amblyopes. This included those for whom this result may have been due to a lack of compliance with treatment. This outcome was defined as a best corrected visual acuity of 6/9 (20/30) or less at the cessation of treatment.

These participants were recruited from a private pediatric ophthalmology clinic. Patients were invited to participate if they had documented evidence of strabismic and/or anisometric amblyopia, had undertaken amblyopia treatment and had been monitored until at least 9 years of age. All participants had ceased amblyopia (or any "maintenance") treatment at least 12 months prior to inclusion and final visual acuity examination. Individuals with ocular disease or manifest nystagmus were excluded from this study. In total, 90 patients satisfied the inclusion criteria and were invited to participate in this study, 42 of which responded positively and constitute our study groups.

For the purposes of this study, amblyopia was defined as an interocular difference in visual acuity of at least 2 lines on the visual acuity eye chart, with the amblyopic eye having a recorded level of visual acuity of 6/12 (20/40) or less. Strabismus was defined as any manifest binocular deviation and anisometropia as an interocular difference in
refraction of at least 1 diopter spherical equivalent between the two eyes.

All participants were examined by a single examiner at what was termed the [study final] followup visit. The examiner was blind to the previously documented visual acuity measurement for each participant. At this followup visit, each participant's best corrected visual acuity was measured using a 3 meter Bailey-Lovie letter chart and a 6 meter Snellen chart, with chart illumination maintained at 150cd/m². The right eye was always tested first, so that consequently the amblyopic eye would be randomly assessed in the order of examination. The participant was asked to read each line in reverse order with the left eye in order to counteract practice effects. The examiner encouraged the participant to read as far down the chart as possible and the visual acuity was taken as the smallest line read with correct identification of 50% +1 of the characters, as reported in the literature (17). Visual acuity was recorded as the reciprocal of the logMAR (log to base 10 of the minimum angle of resolution for the given optotype), and included credit for every letter read correctly, to allow finer differentiation between acuity scores and to facilitate statistical analysis (18). The interocular score was then calculated by subtracting the visual acuity score of the amblyopic eye from the non-amblyopic eye. This interocular score was calculated for two points in time, at cessation of amblyopia treatment and at this [study final] followup visit. The type of vision chart used for scoring and analysis was kept constant over time. For example, if the chart used at the cessation of treatment was a Snellen chart, the same chart was used to measure visual acuity at the long-term followup visit. A two-way mixed analysis of covariance (ANCOVA) was used for these statistical evaluations. The mean period since cessation of treatment was chosen as the co-variant so as to reduce any variability introduced by the time of the followup period. The results were considered "statistically significant" (by convention) when the p (probability) value was ≤0.05.

To detect the presence of strabismus, a [single] cover test was performed for near and distance using an accommodative target. Information regarding the participant's age and visual acuity at commencement of amblyopia treatment, age and visual acuity at cessation of amblyopia treatment were obtained from medical records. The Student t-test and a chi-square were used to analyze the data. Results were considered "statistically significant" (by convention) when the p (probability) value was ≤0.05.

RESULTS

Forty-two participants (n=42; 26 females, 16 males) with a mean age of 14.8 years (range 10-25 years) were examined. Twenty-six participants were considered to be successfully treated (Group 1) and 16 were considered unsuccessfully treated (Group 2).

Fifty percent of the participants in Group 1 and 75% of those in Group 2 had strabismic amblyopia. The remaining 50% of participants in Group 1 and 25% in Group 2 had mixed (strabismic and anisometropic) amblyopia, and none had anisometropic amblyopia alone (that is, without at least microtropia). There was no "statistically significant" difference in the type of amblyopia between the two groups (x²(1) = 1.637, p=0.201 [Yates' correction]).

Table 1 below shows the mean age of the participants, period of followup since cessation and duration of treatment. The participants in Unsuccessful Group 2 were "statistically significantly" older (t[21.5] = -3.194, p=0.004) and had "statistically significantly" longer periods (and later times and ages) of followup than those in Successful Group 1 (t[24.4] = -2.853, p=0.009). The overall mean followup for all participants was 6.6 years. There was not a "statistically significant" difference for mean duration of treatment between the two groups ([40] = 0.599, p=0.553).

At long-term [final study] followup, 62% of participants in Group 1 and 62% of participants in

Table 1. Mean and standard deviation values for age at follow-up visit, period since cessation and duration of therapy for Groups 1 and 2.

<table>
<thead>
<tr>
<th></th>
<th>Mean age at follow-up (ys) ± SD</th>
<th>Mean period since therapy cessation (ys) ± SD</th>
<th>Mean duration of therapy (ys) ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1, n = 26</td>
<td>13.09 ± 2.99</td>
<td>4.68 ± 3.59</td>
<td>4.81 ± 1.78</td>
</tr>
<tr>
<td>Group 2, n = 16</td>
<td>17.55 ± 5.06</td>
<td>8.80 ± 5.04</td>
<td>4.43 ± 2.18</td>
</tr>
</tbody>
</table>
Group 2 demonstrated deterioration in their amblyopic eye visual acuity since cessation of treatment (see Figure 1, above). In Successful Group 1, 23% showed no change in visual acuity and the remaining 15% had an apparent improvement in visual acuity of the amblyopic eye at the time of the [final study] followup visit. In unsuccessful Group 2, 19% had no change in visual acuity and the remaining 19% had improvement.

Group 1: Successfully Treated Amblyopia

In this group, at cessation of occlusion treatment, the mean logMAR visual acuity scores for the amblyopic and non-amblyopic eyes were 1.04 (6/75 + 2 letters) and 1.19 (6/4.8 -0.5 letter), respectively. When comparing the mean VA scores over time, there appears to have been minimal change; the amblyopic eye declined by 1 letter and the non-amblyopic eye improved by 1.5 letters.

The mean interocular score at cessation of treatment was 0.12 (1.2 lines), while at the long-term [study final] followup visit it was 0.20 (2 lines) (See Table 2, below). The interocular score indicates a post-treatment decline in visual acuity at the [study final] followup visit of approximately 0.08 (0.8 lines, representing 4 letters).

Group 2: Unsuccessfully Treated Amblyopia

In Group 2, at cessation of occlusion treatment, the mean logMAR visual acuity scores for the amblyopic and non-amblyopic eyes were 0.72 (6/15 +2 letters) and 1.09 (6/6 -0.5 letters), respectively. At long-term [study final] followup, the mean logMAR visual acuity scores for the amblyopic eye were 1.05 (6/75 +2 letter), respectively. When comparing the mean VA scores over time, there appears to have been minimal change; the amblyopic eye declined by 1 letter and the non-amblyopic eye improved by 1.5 letters.

Table 2. Mean and standard deviation values for monocular visual acuity and interocular scores at cessation of therapy and at follow-up visit for Group 1.

<table>
<thead>
<tr>
<th>Group 1, n = 26</th>
<th>Mean VA ± SD</th>
<th>Mean VA ± SD</th>
<th>Mean Interocular score ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>amblyopic eye</td>
<td>non-amblyopic eye</td>
<td>score ± SD</td>
</tr>
<tr>
<td>Cessation of therapy</td>
<td>1.04 ± 0.05</td>
<td>1.19 ± 0.08</td>
<td>0.12 ± 0.09</td>
</tr>
<tr>
<td>Follow-up</td>
<td>1.02 ± 0.10</td>
<td>1.22 ± 0.09</td>
<td>0.20 ± 0.1</td>
</tr>
</tbody>
</table>
and non-amblyopic eyes were 0.68 (6/15 -1 letter) and 1.12 (6/6 +1 letter) respectively. Again, as for Group 1, when comparing the post-treatment mean visual acuity scores over time, there appears to have been minimal change; the amblyopic eye visual acuity declined by 3 letters and the non-amblyopic eye improved by 1.5 letters.

The mean interocular score at cessation of treatment was 0.37 (3.7 lines). The mean interocular score at the long-term followup visit was 0.45 (4.5 lines) (See Table 3, above). The [study final] interocular score for the amblyopic eye indicates a decline in visual acuity at the followup visit of approximately 0.08 (0.8 lines, again representing 4 letters).

**Comparison of Groups 1 & 2**

Overall, unsuccessful Group 2 showed a larger mean interocular score (difference) than Successful Group 1 at both cessation of amblyopia treatment and at the long-term [study final] followup visit (see Figure 2, below). Both groups achieved an increase in the interocular score over time; this approached, but failed to reach, "statistical significance" for either

### Table 3. Mean and standard deviation values for monocular visual acuity and interocular scores at cessation of therapy and at follow-up visit for Group 2.

<table>
<thead>
<tr>
<th></th>
<th>Mean VA ± SD</th>
<th>Mean VA ± SD</th>
<th>Mean Interocular score ± SD</th>
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<tbody>
<tr>
<td><strong>Group 2, n = 16</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>amblyopic eye</td>
<td>0.72 ± 0.16</td>
<td>1.09 ± 0.13</td>
<td>0.37 ± 0.20</td>
</tr>
<tr>
<td>non-amblyopic eye</td>
<td>1.12 ± 0.14</td>
<td>1.12 ± 0.14</td>
<td>0.45 ± 0.26</td>
</tr>
<tr>
<td><strong>Cessation of therapy</strong></td>
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<tr>
<td></td>
<td>0.68 ± 0.23</td>
<td>1.12 ± 0.14</td>
<td>0.45 ± 0.26</td>
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<tr>
<td><strong>Follow-up</strong></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>0.68 ± 0.23</td>
<td>1.12 ± 0.14</td>
<td>0.45 ± 0.26</td>
</tr>
</tbody>
</table>

Figure 2 (Garoufalis et al): Changes in mean interocular score over time for Group 1 (n=26) and Group 2 (n=16). Time 1 = cessation of therapy, Time 2 = followup visit.
group ($F[1,39] = 3.361, p=0.074$). There was no "statistically significant" difference in the interocular score over time between the two groups ($F[1,39] = 0.031, p=0.860$).

**DISCUSSION**

Sixty-two percent of participants in both Groups 1 and 2 demonstrated a decline in visual acuity of the amblyopic eye at the [study final] followup visit. This concurs with earlier published studies, which have reported that more than half of successfully treated amblyopes will show visual deterioration in the amblyopic eye after cessation of treatment (5,7,9). The amount of visual deterioration over time was, in this study, less than one line on the logMAR chart. These findings are similar to those reported by Ching et al (4) and Rutstein & Corliss (13), but are less than those reported by other earlier studies (9,12). It is noteworthy, however, that these other earlier studies included participants that had ceased monitoring vision prior to 9 years of age, and children whose monitoring ends before this age have been reported to show a greater deterioration of vision in the amblyopic eye(7).

A visual acuity deterioration of less than one line was neither "statistically significant" nor, in our estimation, clinically significant. The small changes in vision in the amblyopic eye observed over time, which included both improvement and deterioration, is most likely due to variable inter-examiner reliability, which has been reported to vary from 0.10 (19) to 0.24 (20) logMAR units. Visual acuity measurements at the cessation of occlusion treatment and at the [study final] followup visit were made by different examiners. The observed greater tendency for the interocular score to increase over time could be due to as simple a matter as the examiner in the past not trying to "push" the participants beyond the 6/6 line. [Sadly, this is an all too common clinical practice anyway. But now that the corneal and refractive surgeons are providing their patients regularly better than 6/6 and 20/20, these visions will no longer be "good enough" and it will become routine to use the 20/15 = 6/4.5 and 20/10 = 6/3 lines on our eye charts -Ed] This possibility and explanation is further supported by the fact that the non-amblyopic eye in both groups showed a mean improvement in visual acuity over time. On closer inspection of the results, 13 out of the 42 cases were recorded to have a level of visual acuity of 6/6 in the non-amblyopic eye at cessation of treatment. This often improved to better than 6/6 at the followup visit. This is an inherent limitation of the retrospective nature of such a study as this is. However, similar testing conditions and identical vision charts were used at both visits to minimize this problem of variability as much as possible.

We also found that both the proportion of patients showing deterioration in amblyopic eye visual acuity and the level of deterioration was similar for the successfully and unsuccessfully treated patients. In contrast, Simons & Preslan (16) reported that untreated or non-compliant patients were significantly more likely to have poorer vision outcomes than those whose amblyopia was treated. Similarly, Haase & Wenzel (15) reported that untreated adults show significantly lower levels of visual acuity as compared to untreated children. They suggested that this is indicative of spontaneous worsening of amblyopia during adolescence. However, both these studies did not specifically compare successful and unsuccessful amblyopia treatment outcomes. Simons & Preslan (16) compared treated and untreated amblyopia, and non-compliant to compliant patients at various visual acuity levels, while Haase & Wenzel (15) only assessed untreated patients. Given that our findings differ from these two studies, it is possible that the stability of vision in non-compliant or untreated patients (which we did not attempt to consider in this study) is different to that of unsuccessfully treated patients. However, this is difficult to conclude definitively by comparison, as some of the patients in our unsuccessfully treated group may have had poor outcomes due to non-compliance rather than resistant amblyopia. Furthermore, the untreated patients in Simons & Preslan's study were under the age of 9 years and only had a followup period of one year, while the cross-sectional nature of Haase & Wenzel's study meant that variables other than amblyopia may have contributed to their findings.

Although neither statistically nor clinically significant, we did find that the UNsuccessfully treated group had a slightly greater level of deterioration than the Successfully treated group. The unsuccessfully treated group had longer followup periods that may have contributed to these findings. However, it should be reiterated that the difference was not "statistically significant" Interestingly, the unsuccessfully treated group also had an over representation of strabismic amblyopia (as opposed to mixed type). This is in contrast to the literature (21) but may be explained by the fact that mixed amblyopia may have been proportionally greater at the commencement of treatment. Some studies have reported that anisometropia decreases with age (22,23). The participants in the unsuccessfully treated group were also significantly older at the followup visit than the successfully treated group, and the diagnosis of strabismic amblyopia may not have always been the same diagnosis due to anisometropia decreasing over time. That is, there may otherwise have been more cases of mixed amblyopia. Previous studies have also shown that patients with mixed amblyopia are more likely to show visual deterioration over time than pure strabismic or anisometropic amblyopes (6,24).

In our study, approximately 23% of the amblyopes
in Group 1 and 19% in Group 2 had an apparent improvement in visual acuity. This improvement rate is lower than those reported by Leiba et al (8) (being 50%), Levartovsky et al (7) (36%) and Ohlsson et al (10) (33%), but greater than the 15% improvement reported by Rutstein & Corliess (13). Levartovsky (7) suggested that improvements in visual acuity were most likely due to practice effects, rather than further therapeutic intervention, given that at long-term followup visits participants were older and better able to cooperate with visual acuity testing. The interocular score was utilized in this study to control for these practice effects. However, this score will only control for practice effects if the improvement in visual acuity is equal between the two eyes. This apparent improvement in visual acuity may also be partly due to inter-examiner bias. Visual acuity scores at cessation of treatment and at the long-term followup visit were determined by different examiners. The method employed to assess visual acuity in the past may have been different to the method applied at the followup visit. A close relationship has been found between visual acuity obtained and the testing criteria applied to assess vision (25); we attempted to reduce this source of variability by using the same type of vision chart and testing distance as previously documented.

It is also possible that participants who were older at the followup visit were more cooperative and more motivated during the examination. In addition, the possibility of an ongoing process of maturation of the visual system cannot be excluded. The development of visual acuity has been suggested to peak in the mid-twenties (19) and this improvement may represent the continuation of this process. Furthermore, other studies have also reported improvements of visual acuity in the amblyopic eye after loss of the good eye in adults (26,27). This suggests that maturation of the visual system may be a gradual process that is not complete at the same stage in all individuals.

Overall, the utilization of the interocular score, instead of analyzing the amblyopic eye alone, allowed a more meaningful interpretation of visual acuity over time. Ching et al (4), who also reported visual acuity as a difference between the two eyes, found a much lower incidence of deterioration, approximately 22%. This lower rate may be due to the their shorter followup period, which was only up until 12 to 13 years of age, and longer monitoring period, up until 10 years of age. However, their study reported a level of deterioration of less than one Snellen line, similar to the findings of our study.

CONCLUSION

Our study results show that visual acuity is essentially stable over a mean followup period of 6.6 years and that amblyopia treatment results in a lasting improvement in visual acuity, regardless of whether treatment was deemed to be successful. Our findings also suggest that the long-term stability of unsuccessfully treated amblyopia is similar to that of successfully treated amblyopia. Amblyopia treatment should therefore be encouraged as even short-term treatment measures and results make a tangible long-term difference in adult populations.

REFERENCES


A PREANNOUNCEMENT:

Extravaganza in New Orleans  
Wright Foundation and Alcon Laboratories

Pre AAO Pediatric Ophthalmology Day  
Course Director: Kenneth W. Wright, MD

Saturday, November 10, 2007 *(Prior to the AAO meeting)*  
Ochsner Institute, New Orleans

Course begins 7:30 am  
Course ends 5:00 pm  
8 hours of CME credit will be offered  
Fee $185.00

Organizing Committee  
Sonal Farzavandi, FRCS(Edin)  
David Granet, MD  
Elias Traboulsi, MD  
Kenneth Wright, MD

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Fax to: 310 - 652-6463 or mail to:  
Wright Foundation, 520 S. San Vicente, Los Angeles,  
California 90048, USA
Strabology Report: 2nd Pediatric Ophthalmology and Strabismus Day
An Extravaganza in Las Vegas, USA, November 10, 2006
Immediately Preceding the American Academy of Ophthalmology Annual Meeting

Meeting Reported by: Sonal Farzavandi, FRCS

Meeting Director: Kenneth Wright, M.D.

Organizing Committee: Sonal Farzavandi, FRCS, David Granet, M.D., Malcolm Ing, M.D., Elias Traboulsi, M.D., and Miho Sato, M.D.

The charm and vibrancy of the fun filled casino city - Las Vegas - attracted 175 pediatric ophthalmologists from 30 countries to attend the pre-AAO Pediatric Ophthalmology and Strabismus Day. This one day meeting, the second of its kind, was hosted by the Wright Foundation for Pediatric Ophthalmology and Strabismus and Alcon Laboratories Inc.

The meeting began with a breakfast symposium on "Pediatric Cataract Surgery Techniques". Following this, there were 3 scientific sessions with free papers on strabismus and 2 strabismus round tables with exciting case presentations and discussion. The pediatric ophthalmology free paper session was held in the afternoon and focused on ocular tumors, ROP, pediatric cataracts and IOL. There were 2 round table sessions: "Ocular Tumors" and "Challenging Cases in Pediatric Ophthalmology"

This report will review only the strabismus papers presented at this meeting.

Scientific Session 1

Richard Gardner, MD et al, from Moorfields Eye Hospital, England, presented a retrospective review of 58 patients who were on long term botulinum toxin injections for strabismus. (Long term defined as having more than 25 injections.) Exotropia was the most common deviation and the lateral rectus muscle was the most frequently injected muscle. He concluded that botulinum toxin injections can be useful on a long term basis in patients who have had multiple previous strabismus surgeries, have poor binocular potential or who are unfit for surgery. The angle of deviation was noted to decrease with time and the interval between injections increased with time. No serious adverse effects were reported with long term use.

An interesting discussion followed with questions on mean time between injections, and if the muscle looked any different during surgery following long term injections. Dr. Gardner stated that the mean time between injections was 6 months over the whole group. The reason for decrease in the angle and increase in the time interval between injections was attributed to the fact that, over time, the fibers that dropped out were mainly the singly innervated fibers. They are important in saccadic induction and power. [Spencer RF, McNeer KW. Botulinum toxin paralysis of adult monkey extraocular muscle. Structural alterations in orbital, singly innervated muscle fibers. Arch Ophthalmol 1987; 105:1703-1711]. Intraoperatively, the muscle looked no different when compared with other patients who have had multiple surgeries.

Dr. M. Salah, from Saudi Arabia, studied the effect of adding inferior rectus recession to inferior oblique recession for correction of esotropia and large V pattern in cases with congenital bilateral superior oblique palsy presenting with a small angle esotropia in primary position. He commented that adding bilateral inferior rectus recession will correct the bilateral fallen eyes, the large V pattern and the small angle esotropia in primary position.

Notable in this session was the paper by Irene Ludwig, MD, from the USA, on trauma causing avulsion-type flap tear of the extraocular muscles in 35 cases.
Etiology included blunt trauma and post-retinal detachment repair. The key to diagnosis is the history, wherein the patients often have symptoms which are far worse than the degree of head trauma. Clinically, it can mimic a pseudo-muscle palsy (tether created by torn flap) or pseudo-entrapment. The direction taken by the flap during healing determined the resultant strabismus pattern. Intraoperatively, one would see a thin and narrow appearance of the attached muscle, lack of capsule or muscle encased in orbital fat. Recognizing the tear and finding the flap is not easy at first. Early repair is advised using braided polyester suture. Worst results were in cases with orbital fracture repair prior to presentation, those with prior attempts at strabismus repair and those with delayed surgery. Best results were obtained with simultaneous fracture and strabismus repair or early strabismus repair alone.

Strabismus Round Table - A

Mary O'Hara, MD, from the USA, presented a rare case of superior oblique palsy in a dysgerminoma survivor. A sixteen year old female with bilateral oophorectomy done for ovarian dysgerminoma presented 4 months post chemotherapy with frontal headache and vertical diplopia. MRI scan was normal. Clinically there was papilledema and a left superior oblique palsy. She was diagnosed to have idiopathic intracranial hypertension. Normalization of the intracranial pressure resulted in resolution of the superior oblique palsy.

This was followed by a magic show by David Granet, MD of the USA. This magic video show even outshined the famous magic shows by his name sake, David Copperfield. He showed a video of Brown's Syndrome "fixed" by exaggerated forced ductions and no surgery. David Guyton, MD was right there to see his forced duction test being used as a cure for Brown's Syndrome. Magic exemplified! This was followed by a video of "magician's forceps", where the contralateral eye moves when the ipsilateral eye is ducted under general anesthesia. This was first reported by Tamura in 1986 [Tamura O, Mitsui Y. The magician's forceps phenomenon in exotropia under general anaesthesia. Br J Ophthalmol 1986; 70:549-552].

The audience was just about recovered from the entertaining magic show when they were challenged by yet another very intriguing case presentation by Ken Wright, MD. This was a 4 year old child who had undergone a superior oblique tendon expander procedure for Brown's Syndrome elsewhere, and developed an overcorrection and consecutive head tilt. Dr. Wright was consulted. He stated that if there is an overcorrection after superior oblique tendon expander, with inferior oblique overaction, inferior oblique recession with partial anteriorization is the procedure of choice. In this case, because of the large head tilt, and minimal inferior oblique overaction, Dr. Wright used an ipsilateral Harada Ito procedure. This worked well as it addressed the extorsion that was driving the head tilt.

David Guyton, MD concluded this session with a case of unusual hyperdeviation in thyroid ophthalmopathy. A young female with thyroid myopathy presented with vertical diplopia due to a right hypertropia with right inferior oblique overaction and right extorsion. Forced duction testing showed a normal left inferior rectus (not tight) and a tight right inferior oblique. No enlarged extraocular muscles were seen on scans. She had a right inferior oblique recession; however, 2 weeks later the symptoms returned and forced ductions showed again diffuse tightness of the right inferior oblique. This was corrected with a right inferior oblique denervation and extirpation.

Ken Wright, MD shared with the audience a similar case he had seen many years ago, where he biopsied the inferior oblique and it turned out to be fibrotic. He concurred with Guyton's findings that the muscle does not necessarily have to be enlarged; it can be contracted due to fibrosis.

Scientific Session 2
Malcolm Ing, MD, surfing champion from Hawaii, USA, presented a retrospective series of 41 patients and a prospective series of 44 patients with congenital esotropia wherein the esotropia deviation showed a progressive increase and thus required an increase in the surgical dosage when compared to the plan made a month or two prior to surgery [Ing MR. Progressive increase in the angle of deviation in congenital esotropia. Trans Am Ophthalmol Soc 1994; 90:117-125; Ing MR, Norcia A, Stager D et al. A prospective study of alternating occlusion before surgical alignment for infantile esotropia: One year postoperative motor results. J AAPOS 2006; 10:49-53]. Dr. Ing stated that the late Dr. Marshall Parks, 3 months before his demise, had commented that this study showed that the medial rectus muscles get progressively tighter with time and this gives us motor reasons to justify early surgery. This was in line with Dr. Wright's prediction several years ago for the need of early surgery for infantile esotropia [Wright KW, Edelman PM, McVey JH, Terry A, Lin M. High grade stereoacuity after early surgery for congenital esotropia. Arch Ophthalmol 1994;112:913-919].

Joseph Demer, MD, PhD, from Los Angeles, USA, gave a passionate talk on techniques in pulley surgery. His talk was illustrated with excellent MRI images showing the three types of pulley pathology that cause incomitant strabismus: (a) Heterotopy where pulleys are misplaced; e.g., inferior heterotopy of the lateral rectus (LR) pulley is associated with a V pattern, alternating hypertropia when bilateral. While early cases were operated by superior transposition of the LR insertion and posterior fixation to the sclera of the superior border of the LR tendon, a more effective operation may be imbrication or tucking of the ligament that joins the LR and superior rectus pulleys. (b) Instability, the gaze related shift of orbital pulleys; similar approaches as described above were advised for LR pulley instability. (c) Inferior rectus (IR) pulley hindrance typically arises from inferior orbital trauma or lower eyelid surgery. Hindrance to IR pulley may be treated by surgical release via transconjunctival inferior orbitotomy, depot steroid injection and inferior traction suturing.

James L. Mims III, MD, from Texas, USA, presented a very interesting study titled "The exoshift under anesthesia at the first and second surgeries reveals that fusion reduces medial rectus hyperinnervation in infantile esotropia". The study addressed the question: Is medial rectus (MR) hyperinnervation among infantile esotropes reduced to normal after successful surgery to align the eyes? Mims and Aaron M. Miller, MBA, MD tabulated measurements of the angle under anesthesia made from 1988 to 1998 using a sterile prism bar and the surgeon's fiberoptic headlight. The difference between the preop' strabismus angle in the awake patient and the angle under anesthesia, the "exoshift under anesthesia", appears to parallel the level of innervation of the medial rectus muscles. From a large series of such measurements, Mims & Miller concluded that the MR hyperinnervation among 17 infantile esotropes was down-modulated by 2/3 after successful horizontal alignment, with normal indicated by a series of 21 patients undergoing surgery for superior oblique palsy, with some residual hyperinnervation to compensate for lateral rectus contracture. Among 27 patients with residual or recurrent esotropia averaging half the original angle preoperatively, previous MR recessions did not result in any reduction in the initial MR hyperinnervation of infantile esotropia. Also, they found that the small residual hyperinnervation required to compensate for contracture of the antagonist LR to maintain orthotropia reverted to normal in 16 consecutive exotropes.

Miho Sato, MD, from Japan, spoke on Class III and Class IV tendon anomalies of congenital superior oblique (SO) palsy. She highlighted that imaging studies were essential for detecting severe tendon anomaly and Class III and Class IV were difficult to distinguish without exploration of Tenon's capsule.

Irene Ludwig, MD commented that she had moved to doing SO advancement
instead of tucks not too long ago. She said "I looked at the tendon in almost every case. I think it's even more common than you think - the more minor anomalous insertions are very common and I see a lot of it in V-pattern esotropia in kids, especially an arrow pattern. So I think you are right but I think these tendon anomalies are more common than you think."

Dr. Wright shared his views on 2 patients who had traumatic avulsion of the SO tendon and presented with SO palsy. Guess what kind of strabismus they had? Almost no vertical deviation in the primary position. They had significant extorsion worse in downgaze and to the opposite side. The vertical deviations were almost not noticeable. If by removing the SO tendon, in an otherwise normal patient, one gets only a small vertical deviation, how do you explain the large vertical deviations seen with congenital superior oblique palsy, based solely on the SO anomaly? It is obviously more than the weak superior oblique and the larger vertical must be due to inferior oblique overaction and/or secondary superior rectus contracture. Dr. Wright emphasized that it's just not a palsy.

Scientific Session 3

David Guyton, MD gave a succinct presentation on "Inverted Brown's Syndrome" due to a tight or inelastic inferior oblique muscle. Twelve patients presented with at least 8 prism diopters of hypertropia in gaze down and in, with superior oblique underaction without ipsilateral inferior oblique overaction. Forced ductions showed that 9 out of 12 patients had tight inferior oblique. None had a lax superior oblique tendon on exaggerated forced ductions. Etiology: 3 had surgery for Brown's Syndrome, 4 had orbital floor fracture repair and 5 had only apparent superior oblique paresis. Four of the 12 underwent contralateral inferior rectus recession and postoperatively all 4 had recurrence of superior oblique overaction over an average followup of 11 months. Eight of the 12 had inferior oblique weakening and had good results. Inferior oblique weakening yields better results than contralateral inferior rectus muscle recession, even though there is no significant inferior oblique muscle overaction preoperatively.

Ken Wright, MD presented a paper on the use of a novel grooved hook for rectus recession under topical anesthesia and for inferior oblique surgery. The grooved hook facilitates exposure of rectus muscles and inferior oblique muscle allowing safe suturing of the muscle directly over the hook for recession surgery. The groove in the hook guides the needle pass, protecting the underlying sclera from inadvertent needle puncture, while providing consistent suture placement with respect to the scleral insertion. It is also useful for suturing inferior oblique muscles as it protects the sclera in the area of the macula. Over 200 muscles were operated upon using this hook. The grooved hook is especially helpful for suturing of tight rectus muscles and for topical anesthesia strabismus surgery wherein the patient will experience pain when the muscle is lifted off the sclera using standard strabismus hooks (Figures A-D, next page). (Dr. Wright has a US patent and a financial agreement with Titan Surgical Company.) He stated that though he has a financial interest in the hook, he can still not quit his day job!

Dr. Wright's talk also had videos of surgery showing the use of the Wright hook for inferior oblique surgery. This has been recently published by Springer in the 3rd edition of the "Color Atlas of Strabismus Surgery, Strategies and Techniques" by Kenneth Wright. This comes with a DVD of 15 strabismus surgical procedures.

Following this talk, Mike Brodsky, MD from Arkansas, USA, presented a paper co-authored with Dr. Wright on three children with infantile esotropia and head oscillations where surgical treatment of the infantile esotropia produced simultaneous resolution of the head shaking/nodding. A
Figure A ABOVE, LEFT: (Wright) - Wright grooved hook. This is made of titanium. The groove is angled towards the handle so when placed under the muscle the groove will be close to the scleral insertion. This facilitates suture placement in the muscle close to the scleral insertion.

Figure B ABOVE, RIGHT: (Wright) - Drawing showing Wright grooved hook under a rectus muscle.

Figure C BELOW, LEFT: (Wright) - Photograph of Wright grooved hook under tight medial rectus muscle. Note that by pulling on the hook the muscle insertion is brought into the surgical field. You suture directly over the groove.

Figure D BELOW, RIGHT: (Wright) - Eye Muscle sutures in place after suturing over the groove in the hook.
video was shown of Dr. Wright's patient with the head nodding preoperatively and its resolution postoperatively. Dr. Wright was using a pen as a fixation target to elicit the eye movements. A timely comment from Ed Wilson, MD who asked if the fixation target used by Dr. Wright was also special and called "Wright's pen" and he was right; it was indeed a "Wright Foundation pen". The audience roared with laughter.

Strabismus Round Table - B
Tony Murray, MD, from South Africa, shared with us a rare case of traumatic strabismus. A young man was stabbed in the forehead resulting in a laceration involving the right upper lid. The incision went through conjunctiva and Tenon's capsule, but spared the globe and the sinuses. The patient had limited adduction in the right eye. CT scan showed that he had a slipped medial rectus muscle. It turned out that, although the anterior part of the muscle sheath and tendon was cut, the posterior part of the sheath was intact, and the intermuscular membrane was spared. It was therefore relatively easy to find the slipped muscle and reattach this to the original insertion. The patient regained full motility. However, as he had an unsightly notch on the upper eyelid margin, he returned two weeks later for further revision, at which time he had sustained a further canalicular laceration on the other side!

Ken Wright, MD and Malcolm Ing, MD each presented a case of overcorrection following inferior oblique recession and contralateral inferior rectus (IR) recession for congenital superior oblique palsy. Ing's case was caused by a stretched scar inferior rectus with the IR slipping back. Wright's case was an over-anteriorized IO with the IO found anterior to the IR insertion. Clinically, the slipped IR has a greater hyper in downgaze and the over-anteriorized IO causes a greater hyper in upgaze associated with limited elevation of the eye with the inferior oblique anteriorization. Wright noted that it is important to avoid anteriorizing the IO anterior to the inferior rectus insertion and to keep the posterior fibers of the IO several millimeters posterior to the anterior fibers thus avoiding the "J" deformity [Guemes A, Wright KW. Effect of graded anterior transposition of the inferior oblique muscle on versions and vertical deviation in primary position. J Aapos 1998; 201-206].

An entire day of intense academic exchange concluded with a wine reception.

The Third Pediatric Ophthalmology and Strabismus Day, again hosted by the Wright Foundation and Alcon Laboratories Inc., will be held on November 10, 2007 in New Orleans, USA, at the Ochsner Institute, immediately preceding the next annual American Academy of Ophthalmology meeting. For inquiries, please contact:
Gabby at gwilliams34@gmail.com or Sonal at sonal@signet.com.sg

The papers and discussion at this meeting were audiotaped and are available from Audio-Digest Foundation. Contact:
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DISCLAIMER: While the reporter has endeavored to be as accurate as possible in reporting the presentations at this meeting, the reader is strongly advised to confirm any information in this report, for sure, before acting upon it or applying it to patients!
Pre AAO Pediatric Ophthalmology Day
Course Director: Kenneth W. Wright, MD

Saturday, November 10, 2007 (Prior to the AAO meeting)
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