KUSHNER: Overaction of the Inferior Oblique Muscle in 4th Nerve Palsy (Correspondence)

*** ORIGINAL SCIENTIFIC ARTICLES ***

DE WIT: Clinical Usefulness of the Aniseikonia Inspector: A Review

MIMS III, AM MILLER and SCHOOLFIELD: The Exoshift Under Anesthesia Correlates with Probable Changes in Medial Rectus Innervation after Surgery for Infantile Esotropia

LEONE, KOKLANIS, GEORGIEVSKI and WILKINSON: Macular and Retinal Nerve Fibre Layer Thickness in Strabismic and Anisometropic Amblyopia

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WANG and C GONZALEZ: Unilateral Myelinated Nerve Fibers Associated with Hypertropia, Strabismus and Amblyopia. "Reverse Straatsma Syndrome?"

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-Watzlawick, 1976

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(Re: Strabology Report of the 34th Annual Meeting of the AAPOS Binocul Vis Strabismus Q 2008; 23:114-128 A letter to Jim Mims III, MD [meeting reporter] and copied to BV&SQ for publication)

Dear Jim:

At the end of your thoughtful review of the 34th Annual Meeting of the AAPOS, you stated that overaction of the inferior oblique (IO) muscle in 4th nerve palsy is due to contracture, and that contracture alone would not explain the hypertropia in contralateral upgaze. You hypothesized that the excyclotropia accompanying a 4th nerve palsy would rotate the insertion of the ipsilateral superior rectus (SR) muscle temporally, putting it on stretch and thus increasing the strength of that muscle. I just re-read your review and noted you ended your hypothesis with the challenge “Prove me wrong.” To that end, I offer these comments:

1. The normal SR has a greater elevating action in abduction than adduction because the muscle’s course makes an angle of about 23 degrees with the A-P axis. When the eye is in abduction the A-P axis is more parallel with the course of the SR making the SR more purely an elevator. The extorsion that occurs with 4th nerve palsy would indeed move the SR insertion further temporal as you suggest. This, however would move the position of maximum elevating force of the SR even further temporally. Thus any increased strength of the SR would be more evident in abduction than adduction. One would expect there to be more of a hyper in ipsilateral upgaze (the field of the SR) than contralateral upgaze (the field of the IO). Thus your hypothesis wold not explain the “overaction of the IO” in its field of action.

2. I do not know if you actually calculated the magnitude of the stretching of the FSR that would occur from torsion, but it is minuscule. If we assume 8 degrees of excyclotropia with a 4th nerve palsy (a typical amount), the equator of an eye with a hypothetical 24 mm axial length would rotate 1.7 mm temporally. If we assume the SR pulley is about 5.6 mm behind the center of rotation, the stretch of the SR would be only 0.2 mm between the insertion and the pulley. This calculation is arrived at by assuming a right triangle with one side 5.6 mm, another 1.7 mm and calculating the hypotenuse as being 5.8 mm. In reality the actual stretch of the SR would be less than 0.2 mm, as the SR inserts anterior to the equator and the millimeters of temporal movement would be less than 1.7 mm as one moves anteriorly on the globe. The math in calculating this solid geometrical construct gets very complex and would be influenced by some variables including how the muscle wraps around the globe. There are also some other assumptions made in the calculation that make it less than precise, all of which would result in stretch that is somewhat less than what I calculated. But in the spirit of being generous to your hypothesis, I will assume the maximum stretch of 0.2 mm, which is quite small indeed.

3. If there were in fact stretching of the SR due to the excyclotropia, its maximum effect should be seen immediately, as sarcomere remodeling should rapidly compensate for any stretch of this small amount. This should negate the effect you are describing. In fact, we see the “overaction of the IO” develop over time.

4. There is a different mechanism by which the extorsion can cause “overaction of the IO” which should not be affected by sarcomere remodeling nor wear off with time. The excyclotropia should move the insertion of the ipsilateral IO nasally. We know the normal IO has a greater elevating action in far adduction as the A-P axis is more parallel with fibers of the IO in adduction. If the insertion of the IO moves nasally, the point of maximum elevating action of the IO will be reached earlier
as the eye rotates into adduction, thus increasing the vertical torque vector for any given position of the adducted eye. This hypothesis, however, would not explain why the “overaction of the IO” increases with time.

5. You stated that I have theorized the ipsilateral IO becomes hypertrophied due to the chronic head tilt with 4th nerve palsy, and that hypertrophy had not been found on imaging studies. As I have said to you in numerous private discussions, I feel the IO develops an increased contractile force which is not synonymous with being hypertrophied. Certainly many strengthened muscles may show hypertrophy, which can be a function of an increase in the number of sarcomeres side by side. However muscle physiology is amazingly complex, and a muscle may show increased contractile force without hypertrophy if there is a change in the ratio of different fiber types, e.g., fast twitch, slow, etc. Picture the athlete who is thin and wiry, yet very strong. (S)he may not have excessive muscle bulk, yet has muscles that are stronger than those of comparable size in another less fit individual. There are also ophthalmologic correlates of this. When the SR Contracture/Overaction Syndrome of Jampolsky occurs idiopathically, the SR is not hypertrophied on imaging, yet clearly behaves like a muscle that generates a stronger contractile force than normal.

I welcome your reply to each of these points.

Burton J. Kushner, M.D.
Madison, Wisconsin

**Practice Opportunity for Pediatric Ophthalmologist-Strabologist**

**Tucson, Arizona.** (An email from Barton L. Hodes, MD) After Brad Schwartz had Brian Stidham “offed” in the fall of 2004, we’ve had a brutal shortage of pediatric ophthalmologists in Tucson. There’s only ONE in private practice, Sam Sato. (Wayne Bixenman, my fallback guy is terrific, but too busy to satisfy the demand.) A pediatric ophthalmologist who is good with people will literally DROWN in patients within months of setting up shop here and will find himself/herself BEGGING for an associate before the end of year #1. The community needs someone and will support the right person to the Nth degree. I’m serving my 4th term on the Medical Executive Committee of the biggest hospital here (Tucson Medical Center) and have the ear of the CEO and CMO.

Barton L. Hodes, MD
Bartonlyle@aol.com

---

**Aniseikonia Question**

Would like the thoughts of the readership.

A 64 year old white male was presented to me February 8. History included: Branch Vein Occlusion, 2 vit, RD with bubble, IOL - all over a period of 5 months - sees 2/15 corrected with -1.00 +1.50 cx 10. Was a -5 D myope and still is in the other eye. I found 3-4 pd left hypertropia managed with 2 pd with “improved” fusion, “not perfect’. Now, adult MD tells him that his diplopia is due to the axial aniso and that IOL surgery on the other eye (20/20 with Rx) would likely solve the diplopia complaints. Thanks.

Miles J. Burke, MD
Cincinnati Ohio
EDITORIAL: Whither Scientific Publication in the Internet Age: Are Books and Hard Copies Going to Disappear? Major Aniseikonia Review; Amblyopia and the Retina; Binocular Motility and Alignment: The Exoshift Under General Anesthesia Revisited; Straatsma Syndrome. Followups on Civility and “System Failure”.

Eight years ago, at the height of the dot.com bubble (remember those unreal better times for which we are paying in spades on margin now, for sure.) your editor made a major donation to an academic library. I had suffered a stroke, knew I had a bad heart valve, was waiting for open heart surgery and was facing and planning for limited longevity. “Dead for sure in two years without it” they said. The surgery, a valve leaflet wedge resection, went very well. They kicked me home on my fourth postop’ day! and now eight years later, my cardiologist says my echocardiogram is still essentially normal!

After I survived a few postop’ problems, which included a thrombosed hemorrhoid due to too much pain medication which I took to sleep because benadryl, the only sleeping med they would give me, didn’t do anything and staying awake all night will drive you crazy; loss, two weeks postop’, of an expensive titanium dental bridge and a cracked tooth from the general anesthesia tube and finally a 6 week postop’ readmission for fainting due to a recalcitrant SVT (supraventricular tachycardia) It was refractory to a full day of medical Rx in the local ER under the direction of my cardiologist. But transporting me to the 4000 foot lower altitude of Denver made it convert. My surgeon thanked me for putting his surgical suture technique to such a thorough test (from a heart rate of 180-190 for twelve hours).

It wasn’t long after all this that I realized that I was probably going to survive ... I thought to myself. regarding my donation: “what the heck did you do that for? In this new internet age, conventional libraries are like hatching dinosaurs. Maybe you should have put the money in a dot.com instead!” But I did have a bunch of personal and ego reasons for doing it. And the internet was just achieving warp speed at that point and the threat seemed not so strong as it has become now, even at our local humble community level, (see adjacent headline) and exacerbated by the current NOT “financial crisis”depression. (See clipping bottom below) more about that later....
BUT I have always loved books and libraries and the hard copy archiving of knowledge and experience (yes, I do keep diaries too). A hard copy means this piece of information is recorded permanently, and forever (especially if on the acid free paper now so commonly available). It is a form of immortality, otherwise unavailable. There is no “delete” command. So you can’t get rid of a hard copy accidentally as is oh so easy on your computer, no? Thank the Heavens for “UNDO”.

We wanted to take this opportunity to look at some of the alternatives now coming available to conventional hard copy scientific publication of which we ourselves have become one by virtue of taking BV&SQ to electronic publication two years ago. BTW, this conversion has gone well except that wrestling with Bill Gates now consumes four times as much time as our old computer programs- WP5.0 which was still all we needed but have finally had to abandon since we can’t convert current word processing programs like Microsoft Office to WP 5.0 or vice versa. (We are using WP 10.0 now. We like “reveal codes”. We do not like flying blind Bill Gates style---he’s crazy). FYI, we do make a hard copy of each issue for archival purposes. We pay the local copy shop to do that for us. Of course any of you subscribers can make such hard copies for yourselves directly from our website for each issue, you can make a copy of it on transportable media and take it to your local copy shop and pay them to make the copy, in black and white or full color depending on your budget.

Our next project for BV&SQ will be to produce an auditory version of each issue with some nice female voice reading it to you page by page, and describing figures and tables as well. Then you can listen to it as you drive to work in your car!!!! or even using an MP3 or similar device, while you read the newspaper in your train seat, double tasking formidably

But let’s look at some other ideas:

The first and most important single question is that regarding OPEN ACCESS to the scientific literature. There is a large portion of the scientific community that doesn’t want to pay anything for scientific journals or the articles usually printed therein. This is usually cloaked in a claim that they are being denied access to the latest (and hottest) scientific information. Based on our having become a full time investor for the past twenty years, [and based on our record would not dare to offer anyone or any of you any investing advice!] We suspect that many members of this demanding community are stock traders who know that breakthroughs, as in medicine and surgery, especially new drug developments can boost the value of a stock immediately and immensely, for those who get the information first (and only for them). Martha Stewart is an example you all know, right?

We and you know there is otherwise little need for this pseudo urgent demand for info and free info at that.

Theoretically there might be some patient who is on death’s door for whom a new discovery might save their life, but we also know that just doesn’t happen.

There is already massive 100% open and FREE access to the scientific literature through the National Library of Medicine and its internet service PubMed which you all know well. The abstracts are free to all and any copies of the full articles can be obtained by hot links for truly nominal fees.

That is not good enough for those who want more. They apparently want 24/7 access to the material pre publication or pre peer approval? or at least some point before anybody else gets it!

But please do read on the next two pages a critical evaluation of the situation by the head of the University Press at Johns Hopkins University and the proposal of the Benham Group, a (?)the leading proponent of pseudo “free” access.
Q: Will university presses survive the Open Access movement to make research available free on the Internet?

A: The short answer is, ‘Yes, of course,’ because the work of university presses is vitally important to academic authors and their readers. Selection, peer review, editing, design, marketing, managing delivery—all are ways that publishers help ensure that an author’s work has maximum impact.

“On the surface, Open Access looks like the library community’s response to the very high prices charged by commercial publishers for science and medical journals. The suggestion is that for those journals, we should move to a system whereby the author would pay a submission fee to the journal publisher out of the grant funding the research. The publisher would use those fees to pay for peer review, editing, design, and distribution, and access would be free. That’s good for library budgets, but are we reducing costs or just moving them from Point A to Point B? If we move to Open Access this way, publishers can no longer sell journal copies or access to people outside academia, like corporate researchers. All funds would come from the academic community. From my perspective, that might ultimately cost the academic community more. Just because information is electronic does not make it free.

“Frankly, I don’t think this approach to Open Access would do harm to university presses, mainly because we are not heavily dependent on publishing science journals. Some have suggested we should publish humanities and social science journals or books in an open electronic manner. It seems unlikely that government funding or private philanthropy will increase enough to permit that, but if it did, university presses would have opportunities to serve our audiences in some interesting new ways. Whatever happens, there will always be a need to select, professionally prepare, and publish the very best scholarship—and to find a way to pay for that important process. We intend to remain a leader in this process, and already have a significant electronic presence through Project MUSE. I think the Open Access rhetoric gets people a little bit worked up, but at the end of the day everyone wants the same thing: a means to produce high-quality, peer-reviewed content and make it widely available. We want to be there to make that happen.”

Kathleen Keane is director of the Johns Hopkins University Press.
September 29, 2008

Dr. P.E. Romano,
PO Box 3727
Dillon CO 80435-3727
United States

Dear Dr. Romano,

Bentham Open are one of the leading international publishers for Open Access journals devoted to various disciplines in science and technology. Please refer to Bentham Open’s website at http://www.oa-opthalmology.org/AllOpenTitles for a current list of publications.

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How about these Bentham Open fees for publication. And you had best become a member too or you may have to wait forever to pay your unaccompanied publication fee.... That highlights one major problem with “open access”. It might be free to the reader but it isn’t free to anybody else, and especially the author and especially if he or she doesn’t have a fat grant or at least one fat enough to pay these various fees!

And that is more of the problem with Bentham’s open access: There is potential for huge conflicts of interest at the publisher. The publisher will want every paper accepted for publication because that’s their source of income. Maybe we could just get along without peer review? Or maybe just not take it too seriously, or demand too much of the author?? He might go to some other open access journal who does not charge so much or demand so much of his academic honesty? Maybe they won’t even have editorial power or peer review. Would you want to do peer review for these open journals? Who will listen to you?

And of course all publishers have similar problems even if they are not trying this freebie route. They are not scientists but all are in business, not academia, primarily and in business to make money, so everything else is secondary to those needs. And strong competition is the only thing that can control the extent to which such conflicts of interest are manifest. But in business, like business owners are more often good friends than fierce competitors- competition is their enemy. No competition, or monopoly or even relative monopoly is more ideal. So as ever, and always. “Caveat Emptor”

Many of you are graduates of Marshall Parks’ training. He practiced and preached private practice and disdained academia, demonstrating the ability of private clinicians to produce meaningful and significant clinical research on their own. Without academic research grants they will have to fund their own publications and all the work to produce it. We went academic but still followed the Parks’ research ideal.
While tracking Bentham down on the web, we ran into a couple of blogs commenting on Bentham’s efforts. One was called Journalsology (cute) and they represented a competitor named BioMed Central. The author accused BPS (Bentham Publishing Services) of lifting some of their web ad material. Another blogger named Nabble had many criticisms of BPS. We did not do a real blog search, however, on BPS.

Here are some more comments on Books and reading per se..

Books Have a Bright Future and Not Just a Digital One

In regard to L. Gordon Crovitz’s “The Digital Future of Books” (Information Age, May 19): Because I have always lived amid books printed on paper and spent a half century or so as a book publisher, my sense of the digital future is different from that of Mr. Crovitz and Jeff Gomez, the author of “Print is Dead” whom he quotes.

The idea that because books can now be stored and transmitted digitally they will necessarily be read on screens is a false inference from incomplete data. Digitization and the Internet will eliminate the traditional supply chain in which physical inventory is stored and shipped to specific retail locations. Instead a multilingual, deep backlist will reside on Web sites of related interest, as well as with aggregators—and be transmitted on demand as swiftly as email to a decentralized world-wide market place where files will be converted to library quality paperbacks by automatic machines—ATMs for books.

Such machines now operate in locations from Egypt to Australia. The machine which we call the Espresso Book Machine because it automatically prints, binds and trims one copy at a time, on demand, quickly for an individual customer is being developed by On Demand Books, of which I am co-founder. Kindles and BlackBerry phones have a place in the digital future. But digitization, the Internet and the Espresso Book Machine will provide readers everywhere the most economical, durable, and ergonomic format yet devised, what Robert Darnton, the Harvard librarian calls the “magic of words as ink on paper” and adds “. . . no computer screen gives satisfaction like the printed page.” or can bring the magic of the digital revolution cheaply and quickly to as many readers.

JASON EPSTEIN
Chairman, On Demand Books
New York

Mr. Epstein was the editorial director of Random House for 30 years. He was also co-founder of The New York Review of Books and founder of The Library of America and The Readers Catalog, the precursor to online bookselling.
JEFFREY P. BEZOS, as chairman, president and chief executive of Amazon.com Inc., made e-commerce mainstream when Amazon started selling books over the Internet in 1994. Since then, he has turned the site into a virtual shopping mall, where the company and thousands of independent merchants sell just about anything from abacuses to zithers. He spoke with The Wall Street Journal’s Walt Mossberg about cloud computing, streaming movies and why books are like horses. Here are edited excerpts of that conversation.

Why the Kindle?

MR. MOSSBERG: You’re doing digital downloads, and hardware with the Kindle [Amazon’s e-book reader]. Why not just stay in your powerful niche of being the go-to place that most people think of as where I buy stuff online?

MR. BEZOS: Most of the extensions we’ve done to our business over time, we’ve questioned ourselves. People thought music and DVDs made sense. But we started selling electronics and toys and apparel and shoes. I’m amazed how many shoes we sell today. It’s so many that we’ve started a focused Web site to just do that.

People understand those product extensions at this point. But [with] new hardware, people look and they say, “What are these guys thinking?” We get there by basing our strategy first and foremost on customer needs instead of what our skills are. It’s important to take an inventory of your skills and try to do things that match up with your skills. But if you only do that, then eventually you will be outmoded because your customers will eventually need things that you don’t have skills for. So you need to renew yourself by developing new skills.

We had been selling electronic books for a long time, and you needed a microscope to find the sales. What customers need is a device that made it very, very frictionless to buy and read electronic books. We wanted to build a seamless, vertically integrated experience, and that required us to develop this whole new skill set.

MR. MOSSBERG: How many have you sold?

MR. BEZOS: We’re not disclosing that. I can give you a new stat that we’ve not shared before. We have 125,000 book titles available for Kindle. When you look at Amazon’s physical book sales of those same titles, the Kindle sales are now more than 6% of those total sales.

ALSO In THIS ISSUE

de Wit GC. Clinical Usefulness of the Aniseikonia Inspector: A Review. Binocul Vis Strabismus Q 2008; 23:207-214. This is the Bible on this subject. Clip for ultimate reference. -per


THE WALL STREET JOURNAL

R4 Monday, June 9, 2008

MR. MOSSBERG: What about the whole idea of people reading on a screen? Just as other kinds of media, like newspapers, see a line of growth for digital and decline for physical, is that going to happen in books?

Jeffrey Bezos

MR. BEZOS: Over some time horizon, books will be read on electronic devices. Physical books won't completely go away, just as horses haven't completely gone away. But there is no sinecure for any technology. If you think about books, it's astonishing. It's very hard to find a technology that has remained in mostly the same form for 500 years. And anything that has stubbornly resisted improvement for 500 years is going to be hard to improve.

That is what we're trying to do with Kindle. We see this as an effort to improve upon the book, even though it's resisted change for 500 years.

To do that, you have to capture the essential element of a book, which is that it disappears when you get into the flow of the story. None of us when we're reading a book think about the ink and the glue and the stitching. All that fades away, and you get into the author's universe.

Sometimes big, heavy hardcover books do break you out of the flow because you get hand fatigue. Or turning pages can be loud if you have a spouse sleeping next to you. There are things about physical books that we're accustomed to but that actually aren't very good.

But you also can't ever outnumber the book. You need to look for a series of things that you can do with an electronic device like Kindle that you could never do with a physical book.

Some of them can be pretty simple, like dictionary lookup. I find I don't know what lots of words mean, and I used to guess because—am I really going to get up off of the sofa and go find a dictionary?

Changing the font size, a very simple thing that's much appreciated.

And then some whoppers. The big whopper is wireless delivery of books in less than 60 seconds. You don't have the cognitive overhead of thinking about your monthly wireless bill. You don't have to know who the wireless carrier is. We're hiding all of that complexity.

MR. MOSSBERG: When I did my Kindle column, I got a lot of email from people who were talking about the tactile feel of the book—the hard-to-describe intangibles around reading a paper book that you lose on an electronic device.

MR. BEZOS: I'm sure people love their horses, too. But you're not going to keep riding your horse to work just because you love your horse. It's our job to build something that is better than a physical book. The reason we love physical books is because we have had so many great experiences with that object in our hands that we have nice associations with it.

We're not trying to displace people's love of that physical object that is the book. It's a hallowed invention. The thing to keep in mind is what's really important is not the container, it's the narrative. Long-form reading is important for our society.

Over the last 20 years, most of the tools that we humans have invented have made it easier for us to be information snackers. If one of the outcomes of Kindle and other devices like it [is] making long-form reading more frictionless so that you end up doing more of it, I think that's a good thing.
Followup: Hostility Replaces Civility

In the last two issues of BV&SQ we have also in these pages bemoaned the loss of civility in our society. Since the last issue, the most impressive examples of this came in all the recent Congressional hearings relating to the so-called “financial crisis.” They call all these prominent people before them and with the TV cameras upon them make great theater criticizing and excoriating these “villains.” What HYPOCRISY heaped upon their on so natural incivility on behalf of their “offended constituents.” The epitome of this unbelievably disgusting display was their harassment of the CEOs of GM Ford and Chrysler for wasting money by flying to Washington in their private airplanes for these ritual Congressional lashings and punishments, when Congress caused this whole mess!

Further he says that no mistakes were made - it was a “system failure.” One of Barack’s new cabinet also said exactly the same thing just a day before Rubin: “NO ONE was at fault, it was a “system failure” There is/are NO guilty parties.

Well the guilt starts with the Head of HUD, Cisneros and Bill and Hillary who joined him in thinking our society needed for everyone to own a house, regardless of their means. So they mandated that, and Congress passed laws forcing the mortgage issuers to give everyone a mortgage regardless of their financial situation. So they did.

That’s where the subprime mess started, and it was quickly compounded by clever Wall Streeters into a variety of highly profitable but totally idiotic securities. These were so faulty that everyone who invested in them has been literally bankrupted by them. And this financial crisis, further exacerbated by other government programs like Fannie Mae and Freddie Mac and the SEC’s mistakes, have resulted in the loss through the 50% devastation of the stock market, not just here, but around the world. And the largest destruction of wealth in the history of mankind. Calling this a “system failure” is likely blaming everything on “computer mistakes” It is rather the most disastrous misdirected government created BUBBLE ever. Human fallibility and incompetence and greed reigns. Mxmas –PER
Clinical Usefulness of the Aniseikonia Inspector: A Review

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from Optical Diagnostics, Culemborg, The Netherlands

ABSTRACT: Purpose: In the literature several articles have appeared which describe the accuracy and repeatability of direct-comparison aniseikonia tests, such as the New Aniseikonia Test and the aniseikonia test of the Aniseikonia Inspector. In these articles a discussion on the clinical relevance of the results is often missing. Therefore, this article discusses the clinical usefulness of direct-comparison aniseikonia tests, in particular that of the Aniseikonia Inspector.

Method: Review of the literature shows that an aniseikonia test is usually evaluated by inducing aniseikonia in normal subjects using size lenses. The range of induced aniseikonia differs. Some investigations evaluated from appr. –8% to +8%, while other investigations evaluate a (too) small range from appr. –3% to +3%.

Results: In general direct-comparison aniseikonia tests have a repeated measurement accuracy of approximately 0.5% standard deviation (this also includes actual aniseikonia fluctuations). A direct-comparison aniseikonia test with free eye rotation seems to underestimate the amount of aniseikonia. However, converting these underestimations into clinically applicable values shows that vertically the ‘error’ is on average smaller than only 0.5% and horizontally the ‘error’ is smaller than 1%. This maybe statistically significant but clinically less relevant underestimation seems to disappear in tests without free eye rotation (as in the newer versions of the Aniseikonia Inspector).

Conclusion: Because aniseikonia does not seem to give any clinical symptoms until the aniseikonia has a value of 3-5%, the accuracy and repeatability of direct-comparison tests, such as that of the Aniseikonia Inspector, is sufficient for effective aniseikonia management.

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INTRODUCTION

Aniseikonia is a condition in which the eyes perceive images of different size. It can either be of optical origin or retinal origin (See Table 1).

Table 1: Possible causes of aniseikonia

<table>
<thead>
<tr>
<th>Optically-induced¹</th>
<th>Retinally-induced²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anisometropia</td>
<td>Epiretinal membrane</td>
</tr>
<tr>
<td>Pseudophakia</td>
<td>Retinal detachment</td>
</tr>
<tr>
<td>Refractive surgery</td>
<td>Macular edema</td>
</tr>
<tr>
<td></td>
<td>Macular holes</td>
</tr>
<tr>
<td></td>
<td>Retinoschisis</td>
</tr>
</tbody>
</table>

If aniseikonia has a retinal origin, it is often associated with the terms micropsia or macropsia. The difference between aniseikonia and micropsia/macropsia is that aniseikonia denotes a difference in perceived image size between the eyes, while micropsia/macropsia often denotes a monocular difference in perceived image size at different moments in time. For example, a patient may experience macropsia in one eye due to an epiretinal membrane: i.e., the image looks larger after the growth of the epiretinal membrane. If the other eye did not grow a similar epiretinal membrane, the macropsia will also result in aniseikonia.

Definition of aniseikonia values
The amount of aniseikonia is defined by the following basic equation¹³:

\[ a \cdot \frac{m_{OD}}{m_{OS}} = 1 \]  \[ [I] \]

with \( a \) the amount of aniseikonia and \( m_{OD}/m_{OS} \) the perceived image size in the right eye relative to that in the left eye. For example, say the images in the right eye are perceived a factor 1.10 times the size of that in the left eye (\( m_{OD}/m_{OS} = 1.10 \)). According to Eq.1, this means that the amount of aniseikonia equals 1/1.10 = 0.91. This example of Eq.1 shows three essentials:
1) aniseikonia is defined relative to the right eye,
2) the amount of aniseikonia shows how much the image in the right would need to be magnified in order to correct the aniseikonia (similar to defining refractive error as the amount of power that needs to be added to correct/emmetropize an eye), and
3) for accurate aniseikonia calculations or evaluations the aniseikonia should be treated as a factor, instead of the more common percentage notation (i.e., the example shows that a +10% image size difference results in a -9% aniseikonia and not a -10% aniseikonia).

Symptomatic aniseikonia values
The aniseikonia values at which a patient becomes symptomatic are important to know, because
- it determines how accurate an aniseikonia test would need to be and
- it determines what range of aniseikonia values to use when evaluating an aniseikonia test.

Binocular functions such as binocular contrast sensitivity, binocular summation, and stereopsis are not affected until the aniseikonia has a value of 3-5%⁴-⁶. The tolerance for aniseikonia in the near periphery seems to be a little higher (6-7%)⁷.⁸. Note that in references 7 and 8 tolerance is defined as the diplopia threshold, while other symptoms may possibly occur at smaller aniseikonia values. Sometimes it is stated that sensitive individuals may also experience aniseikonia symptoms at values from 1-3%⁹. However, based on the objective binocular functions and near periphery aniseikonia tolerance as described above, it seems more likely that these patients are bothered by anisophoria instead of aniseikonia (anisophoria = heterophoria that varies with the direction of fixation). Note that part of the reason why anisometropes often have less symptoms with contact lenses than glasses might be due to the fact that there is no optically-induced anisophoria with contact lenses.

In conclusion, this article will assume that patients do not become symptomatic until the aniseikonia reaches a value of 3-5%.

Interest in aniseikonia testing
Aniseikonia was first studied extensively in the 1940s and 1950s¹,³ but interest in this binocular vision anomaly disappeared again. The main
reasons appear to be: 1) a lack of affordable and easy-to-use (both for the administrator and the patient) test equipment and 2) the difficult or extensive calculations involved in designing an aniseikonic Rx. Today the interest for aniseikonia is reviving again, probably due to a) the introduction of commercially available aniseikonia tests such as the New Aniseikonia Test (NAT) and more recently the Aniseikonia Inspector; b) the fact that iseikonic lenses are easier to design with the advent of computers and the Aniseikona Inspector software, and; c) the realization of the significant and increasing number of aniseikonia patients. To demonstrate the large and increasing number of aniseikonia patients, often, as an example, the pseudophakia group (both unilateral and bilateral) is mentioned of whom approximately 40% appear to have symptoms referable to aniseikonia.

**Article objective**

In the literature several articles have appeared that describe the accuracy and repeatability of direct-comparison aniseikonia tests. In these articles a discussion on the clinical relevance of the results is often missing or conclusions are drawn prematurely. Therefore this article reviews these studies with an emphasis on the clinical usefulness of direct-comparison tests, in particular the aniseikonia test of the Anisekonia Inspector. Also some improvement suggestions are made for the method and analysis of the evaluation studies.

**METHOD**

The way an aniseikonia test is usually evaluated, is by inducing an image size difference in normal subjects with size lenses (lenses without power, but with an optical magnification). In general the derivative (slope) of the dependence between the measured amount of aniseikonia and the induced amount of magnification (both as percentages) is determined to evaluate the “validity” of the aniseikonia test. With a slope statistically different from -1.00, the usefulness of the test has been questioned. Even though determining the slope provides more accurate information than taking the ratio between measured and induced aniseikonia, this article will show that finding a statistically significant difference from minus 1.00 does not mean that the test is not clinically useful. To determine the clinical usefulness, the maximum ‘error’ between the measured aniseikonia and the induced aniseikonia should be established. If this ‘error’ is much smaller than symptomatic aniseikonia values (3-5%), then the test is still useful.

This article will also discuss some improvements that could be made to the method/analysis used in some of the reviewed articles. These improvements could even change the outcome of the statistical analysis in some cases. In short, the improvements are:

- Test for a range of aniseikonia values larger than -5% to 5%.
- Test in a darkened room.
- Test with a fixation disparity correction.
- Analyze based on the true theoretical slope, which may be different from -1.00.
- Analyze using correct size lens magnification values. For example, using a +5% size lens to also minify the image, gives a -4.76% minification and not a -5% minification.

**RESULTS**

Table 2, next page, shows the main results of different articles that evaluated direct-comparison aniseikonia tests. There are basically four different types of tests evaluated:

- **An. Insp. 1**: Aniseikonia Inspector, version 1 (commercially available software showing the direct-comparison targets on the screen).
- **An. Insp. ‘2’:** A prototype research version of newer versions of the Aniseikonia Inspector.
- **NAT:** New Aniseikonia Test (commercially available product showing the direct-comparison targets in a booklet).
- **SDCT:** Self developed computer test (non-commercially available tests).

Basically, all of these tests are similar in that the patient has to choose a pair of size targets (viewed haploscopically with red-green glasses) that are perceived as equal in size. The Aniseikonia Inspector ‘2’ could be considered slightly different in that test is based on a forced choice procedure...
with short exposures to avoid any interference from anisophoria and head movements.

In some studies the sign of aniseikonia was taken opposite to the standard definition as laid out in the introduction. This resulted in slope values close to +1.00 instead of -1.00.

The maximum slope-related ‘error’ is the maximum difference between the measured aniseikonia and the expected aniseikonia over the range of aniseikonia values tested. The word error is in between quotes, because the underestimation might also represent a limit of adaptability. In that case the underestimation might even be useful, since correcting less anisikonia will in general yield cosmetically more acceptable glasses.

The maximum slope-related ‘error’ is the maximum difference between the measured aniseikonia and the expected aniseikonia over the range of aniseikonia values tested. The word error is in between quotes, because the underestimation might also represent a limit of adaptability. In that case the underestimation might even be useful, since correcting less anisikonia will in general yield cosmetically more acceptable glasses.

The last column of Table 2 shows the repeated measurement accuracy. This represents the standard deviation of multiple measurements on the same subject. Some articles do not provide the value of this parameter directly. However, sometimes the researchers did determine the coefficient of repeatability, which is defined as 2 times the standard deviation of the difference between repeated measurements. This repeatability coefficient indicates the maximum difference that is likely to occur between two measurements (95% level). It should be noted that the spread in values of the difference between two measurements is \( \sqrt{2} \) times larger than the spread in values of the measurement itself. Converting the repeatability coefficient (RC) into the standard deviation of the measurement itself (sd) is therefore done with:

\[
sd = \frac{RC}{2\sqrt{2}}
\]

It should be noted that the silent assumption that the aniseikonia is stable over time does not seem to be true. Reasons for these fluctuations could be, for example, fluctuations in adaptation (cortical processing), refraction, or accommodation. So, part of the measurement inaccuracy may also be caused by actual aniseikonia fluctuations.

All data of Table 2 are reasonably consistent, except for one unexplainable outlier (McCormack et al., evaluating the NAT). Not including this outlier, the main results from Table 2 are:

1. The repeated measurement accuracy is in the order of 0.5%.
2. The maximum slope-related ‘error’ is less than approximately 0.5-1%, independent of the range of values that were evaluated.
DISCUSSION

Looking at the results and taking into account that aniseikonia does not become symptomatic until it has a value of 3-5%, it is obvious that direct-comparison aniseikonia tests are very useful for aniseikonia management. A similar conclusion was also drawn by Yoshida et al.\(^2\) and Awaya et al.\(^\)\(^2\)\(^5\). Nevertheless, it might be good to look at the method and analysis of the reviewed articles and discuss some issues that might be improved:

**Testing range**

Table 2 shows that some of the published articles tested only a relatively small range of aniseikonia values (3-4%), while others tested a larger range (8-9%). Since aniseikonia does not seem to become symptomatic until 3-5%, it makes more sense to test for a range that is larger than 5%.

Testing only a range of 3-4% also does not reveal that the slope seems to become closer to minus 1.00 for larger and more symptomatic aniseikonia values (See Fig. 1). Due to this effect, Table 2 shows that the maximum slope related 'errors' do not increase with the amount of induced aniseikonia, but stay less than approximately 0.5% vertically and 1% horizontally.

**Room illumination**

If there are binocular visible objects/texture/shadows in the peripheral field of vision while doing a direct-comparison aniseikonia test, it has been hypothesized that this may result in an underestimation of the measured aniseikonia\(^1\),\(^1\),\(^2\),\(^6\).

If an aniseikonia test requires external illumination (such as with the NAT), the visibility of peripheral binocular objects/texture/shadows might be difficult to eliminate. However, with self-luminous tests this might be achieved more easily. The direct comparison eikonometer described by Ogle\(^1\), projects an image on a screen and peripheral fusion stimuli, for example from the contours of the screen, are eliminated by unlike aperture (a circle and a square) before the eyes. Similarly, the instructions of the Aniseikonia Inspector are to test in a darkened room and the image on the display is such that one eye sees the normal rectangular border of the screen and the other eye sees an artificial elliptical border. Rutstein et al.\(^1\) and Fullard et al.\(^\)\(^1\)\(^9\) also show that a darkened room results in slightly higher slope values. However, clinically these differences are minor and, as mentioned above, maybe the underestimation is even useful as it possibly reveals a limit of adaptation. Nevertheless, when evaluating a direct-comparison aniseikonia test, it is advised to measure with dimmed room illumination (if possible).

**Fixation disparity correction**

If there is a fixation disparity (or heterophoria) during the aniseikonia testing, the two size targets will be misaligned making it more difficult to compare their sizes. This may especially happen in the horizontal direction. To minimize the loss of accuracy due to fixation disparities, the Aniseikonia Inspector software includes the ability to compensate...
for fixation disparities. Some of the studies presented in Table 2 did not make use of the possibility to compensate for fixation disparities. For the most accurate results, it is advised to use this option. In the newest version of the Aniseikonia Inspector, each patient will also start with a fixation disparity test before starting the actual aniseikonia test.

Slope
It is often stated that an ideal test should have a slope of -1, meaning that for each percent image magnification difference increase, the measured aniseikonia should decrease by one percent. This is not completely accurate though. As described by de Wit\(^\text{12}\), the dependence between aniseikonia (A) and the induced image size difference (M) when described as percentages is:

\[
A(M) = -\frac{M}{1 + \frac{M}{100}} + A_0
\]

with \(A_0\) the inherent aniseikonia of the subject, and

\[
slope = \frac{dA(M)}{dM} = -\frac{1}{(1 + \frac{M}{100})^2}
\]

Equation 3 shows the effect described in the introduction that a +10% image size difference results in a -9% aniseikonia. The slope is only exactly -1 for \(M=0\%). For a range of induced image size differences between the eyes that are approximately symmetric around \(M=0\), the slope of a linear regression will also be close to -1.00. For example, the range used by de Wit\(^\text{12}\) (-7.6...8.6%) would give an ideal linear regression slope of minus 0.995. However, the ideal slope for the image size differences used by Ugarte\(^\text{13}\) (0..8%) is -0.926!

Accuracy of size lenses
In most articles the slope is described with a precision of 2 decimal places. For example, the slope might be 0.88 (also sometimes confusingly described as a 12% underestimation). Since the analysis usually does not include any error estimation for the size lens magnification values, it would be assumed that the size lens magnification values are also known with a precision of approximately 2 decimal places. For example, in some articles size lenses are used of said 1%, 2% and 3.5% magnification\(^\text{18,19}\) which then could be assumed to be as precise as 1.00%, 2.00% and 3.50%. This, however might not be realistic when considering the minor fabrication or measurement errors necessary to change the magnification value by a significant amount from the assumed magnification value (see Table 3). To fabricate or even measure lenses with a 0.04 mm center thickness tolerance or a 0.02D residual power tolerance would be difficult to do.

The data of Table 3 was obtained using the ray-tracing tool Zemax using the Gullstrand-LeGrand eye model and a 3% size lens with the following data: material CR-39, front radius 50 mm (9.96D), center thickness 4.32 mm, zero power, a vertex distance of 13 mm, wavelength of 550 nm, and field angle of 4 deg.

<table>
<thead>
<tr>
<th>Fabrication/measurement error</th>
<th>3.00% - 2.97% (slope = 0.99)</th>
<th>3.00% - 2.85% (slope = 0.95)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lens center thickness</td>
<td>-0.4 mm</td>
<td>-0.18 mm</td>
</tr>
<tr>
<td>Base curve</td>
<td>-0.10 D</td>
<td>-0.48 D</td>
</tr>
<tr>
<td>Refractive power</td>
<td>-0.02 D</td>
<td>-0.09 D</td>
</tr>
</tbody>
</table>

Also when ordering size lenses from a company, it is important to verify that the ordered magnification values are even close to the values ordered. For example, Lilja\(^\text{14}\) describes that the 5% size lens they ordered, appeared to be a 3.5% after measuring the curve and center thickness. Also Antona et al.\(^\text{16,17}\) appeared to have received a 3% and 1.5% size lens, instead of the requested 4% and 2%.

Using size lenses to magnify and minify
In most of the reviewed studies the size lenses are used to induce aniseikonia of both positive and negative sign. To change the sign of the
magnification of the size lens either the lens is flipped or the lens is held in front of the other eye. Except in the study of de Wit\textsuperscript{12} the value of the magnification is kept constant for the magnifying or minifying state. This, however, is a flaw in the analysis. The more exact way of calculating the magnification/minification change of a size lens is to treat the value as a factor. For example, a +5.00% size lens magnifies by a factor of 1.0500. By flipping this lens (or switching to the left eye) the minification relative to the right eye becomes 1/1.0500=0.9524 or -4.76%. Even though the difference between -5.00% and -4.76% might not be clinically important, it again could have a large impact when doing a statistical ‘validity’ analysis on a parameter like the slope.

CONCLUSION

The measurement accuracy of direct-comparison aniseikonia tests had a standard deviation of only 0.5% (even including natural fluctuations in aniseikonia). This is much smaller than the aniseikonia values at which a patient becomes symptomatic (3-5%). There appears to be some underestimation in the measurements, but this is on average smaller than only 0.5% vertically and 1% horizontally, both independent of the range of aniseikonia values tested. Because of this independence the underestimation might represent a kind of limited adaptation due to peripherally visible fusion cues and as such could possibly be treated as a useful minimum correctable amount of aniseikonia. Another possible reason for the underestimation could be the involvement of minor head movements during the testing causing prism effects. The newer versions of the Aniseikonia Inspector also have a short-duration stimuli mode, in which the underestimation seems to disappear.

The usefulness of a direct-comparison aniseikonia test should not be based on a possible statistical significant deviation from a slope value of -1.00. Even if there is a statistical significant deviation this article shows that the deviation does not compromise the clinical usefulness. The discussion section of this article also offers several suggestions to improve the method and analysis of the studies evaluating a direct-comparison aniseikonia test.

In conclusion, direct-comparison aniseikonia tests and in particular the aniseikonia test of the Anisokonia Inspector provide sufficient accuracy to be clinically useful.

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The Exoshift Under Anesthesia Correlates with Probable Changes in Medial Rectus Innervation after Surgery for Infantile Esotropia

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ABSTRACT: **Purpose:** To study the outward deviation of the eyes (exoshift) under anesthesia, in a variety of clinical settings in order to improve our understanding of how medial rectus recessions change alignment and innervation.

**Methods:** Pre-operative and intraoperative eye deviations were measured before surgery and under Stage 3 of general anesthesia using a modified Krimsky test in 5 groups: 1) Unoperated infantile esotropia (N = 60); 2) Undercorrected infantile esotropia (N = 27); 3) Corrected infantile esotropia with subsequent vertical deviations (N = 17); 4) superior oblique palsies without horizontal deviations (N=21); and 5) late consecutive exotropia (N=16).

**Results:** Group 2 averaged half the esotropic deviation of Group 1 (19.8 ±7.4 ET vs. 42.1 ±18.3 ET, but had a nearly identical exoshift (41.9 ±13.2 vs. 41.8 ±13.6, p = 0.96). Group 4 (orthotropic) and Group 5 (exotropic) demonstrated smaller and nearly identical exoshifts (26.0 ±8.3 vs. 24.0 ±9.3, p = 0.50). Group 3 had significantly less exoshift (30.1 ±6.0) than Groups 1 and 2 (p = 0.002 for both), but significantly more exoshift than Group 4 (p = 0.04) or Group 5 (p = 0.067)

**Conclusion:** Contracture of the lateral rectus reduced the deviations after undercorrecting surgery, but the exoshift remained unchanged. Medial rectus recession by itself has no effect on medial rectus innervation. Successful surgery substantially reduced the exoshift, but not to the normal level seen in consecutive exotropia. These data combined with a reasonable set of assumptions regarding the state of contracture (expansion) of the horizontal recti in a variety of pre-and post-operative settings lead to the conclusion that setting the eyes straight with a successful bilateral medial rectus recession reduces the pre-operative hyperinnervation of medial rectus almost to normal, with a small amount of residual hyperinnervation remaining to overcome the increased contracture of the lateral rectus which occurs due to increased exotropia during sleep after a bilateral medial rectus recession. When this small residual hyperinnervation decreases to normal, consecutive exotropia develops, owing to continued increased contracture of the lateral recti.

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INTRODUCTION

The Pediatric Ophthalmologist who has the privilege of performing surgery on large numbers of infants with esotropia is quickly impressed with the fact that if orthotropia is achieved in the early days after surgery, the infant usually maintains this good result for months or years. This phenomenon has traditionally been labeled "motor fusion". (See Duke-Elder (1) and von Noorden (2).) Careful measurements of the ocular deviation under anesthesia, which have not proven useful in deciding how many millimeters to recess the medial recti in these infants (3), might yet provide insight into the mechanism of motor fusion, especially when compared to the angle in the awake infant in a variety of pre-operative clinical settings. One of the central questions to be answered in advancing our understanding of how bilateral medial rectus recessions cure esotropia is whether the medial rectus muscle preoperatively is abnormally hyperinnervated and whether this hyperinnervation resolves after the eyes are straightened. Is there a permanent balance between continuing hyperinnervation and a permanent weakening produced by recessions or does the level of innervation of the medial recti become normal after the eyes are straightened? The level of innervation of the medial recti in patients with infantile esotropia cannot be directly measured, but the exoshift under anesthesia, defined as the difference between the angle in the awake patient and the angle under anesthesia, can be measured. A major purpose of this study is to explore whether it is reasonable to equate the exoshift under anesthesia with the level of innervation of the medial recti in the awake patient just prior to the surgery.

METHODS

This retrospective chart review of office charts from the private practice office of the primary author was approved by the Institutional Review Board of the Methodist Healthcare System of San Antonio, Texas, and all data collection was in compliance with the United States Health Insurance Portability and Accountability Act of 1996.

The exoshift under anesthesia, defined as the difference between the angle in the awake patient and the angle at stable Stage 3 of general anesthesia without paralyzing agent, was measured (see Figure 1, next page, below or right) at the start of each surgery in 60 patients requiring two surgeries to correct horizontal or subsequent vertical deviations associated with infantile esotropia and 21 patients requiring a surgery to correct vertical deviations due to superior oblique palsies who had no significant horizontal deviations.

Stage 3, 12 to 15 minutes after the induction of general anesthesia, is the usual stage during which strabismus surgery is performed and is well documented to be associated with eyes that not directed upwards and are no longer moving (4). The angle in the awake patient (as in Figure 1A) was measured with standard alternate cover test with prism for neutralization of movement. The angle under anesthesia was measured using a sterile Berens prism bar and the surgeon's fiberoptic headlight (modified Krimsky technique). As pointed out by Guyton (5), the corneal reflex produced by the surgeon's fiberoptic headlight is usually symmetrical within the pupils of the left and right eyes. (See Figure 1 B.) The surgeon's head was shifted slightly to the right to make the corneal reflex appear normal (normal angle kappa) in the patient's left eye. (See Figure 1 C). Then, a sterile prism bar was placed before the patient's right eye and moved up and down until the corneal reflexes appeared symmetrical. (See Figure 1 D.)

The 60 patients with infantile esotropia receiving two surgeries were classified into four groups, with Group 1 representing the exoshift data observed at the start of the first surgery for all 60 patients, Group 2 comprising the data observed before the second surgery for 27 patients who were undercorrected for esotropia after the first surgery (9 ET or more), Group 3 comprising the data observed before the second surgery for 17 patients with corrected horizontal deviations (5 ET to 5 XT) receiving a second surgery for only a vertical deviation (Dissociated Vertical Deviation and/or overaction of the inferior oblique), and Group 5 comprising the data observed before the second surgery for 16 patients who had late consecutive exotropia (8XT or more). All of the patients in Group 5 were 5ET to 5XT at 6 weeks after the initial surgery, but had developed late consecutive exotropia prior to the second surgery. The 27
patients in Group 2 had residual esotropia of 9 to 40 ET averaging 19.8 ± 7.4 ET. The 17 patients of Group 3 were 5 ET to 5 XT after their initial surgery of bilateral medial rectus recessions and averaged 0.3 ± 2.7 XT awake just prior to their second surgery being performed for only a vertical deviation, inferior oblique overaction or Dissociated Vertical Deviation. The 16 patients consecutively exotropic at the second surgery in Group 5 had exotropic angles of 8 to 35 XT averaging 13.5 ± 6.8 XT. Although each patient in Groups 2, 3, and 5 was also included in Group 1, the awake angles prior to the initial bilateral medial rectus recession were statistically uncorrelated with the awake angles prior to the second surgery. Group 4 comprised the data observed before the first surgery performed for the 21 patients with superior oblique palsies who had no horizontal deviation, with average pre-op horizontal deviation of 0.1 ± 2.0 XT.

Summary group results were reported using means and standard deviations. Group mean comparisons were performed using one-way analysis of variance and unpaired and paired Student's t tests when appropriate. Owing to the small sample sizes in Groups 2, 3, 4, and 5 and the tendency for the data not to satisfy the assumptions of normality, analogous non-parametric tests including Kruskal-Wallis, Mann-Whitney, and Wilcoxon signed ranks were also performed. SPSS 15.0 was used to perform the statistical analysis.

These data were gathered naively and without bias in the sense that the original purpose of
Table 1. RESULTS: Group Characteristics

<table>
<thead>
<tr>
<th>Group Number</th>
<th>Description of Group</th>
<th>Pre-op Awake</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. N = 60</td>
<td>Prior to any Surgery</td>
<td>42.1 ± 18.3 ET</td>
</tr>
<tr>
<td>2. N = 27</td>
<td>Undercorrected (9 ET or more) after MROU</td>
<td>45.0 ± 18.5 ET</td>
</tr>
<tr>
<td>3. N = 17</td>
<td>Ortho (5ET to 5 XT) after MROU</td>
<td>41.3 ± 20.0 ET</td>
</tr>
<tr>
<td>4. N = 21</td>
<td>Surgery performed for Superior Oblique Palsy, no horizontal Deviation, “normal” MR innervation</td>
<td>0.1 ± 2.0 XT</td>
</tr>
<tr>
<td>5. N = 16</td>
<td>Consecutive Exotropia (8 XT or more) after MROU</td>
<td>37.9 ± 16.3XT</td>
</tr>
</tbody>
</table>

Note: One-way analysis of variance of the pre-op awake angle prior to the first surgery for groups 2, 3, 5 revealed no significant difference (p = 0.476).

ET = esotropia, XT = exotropia, MROU = bilateral medial rectus recession

recording the intraoperative deviation at the start of the surgery was an attempt to correlate the results of the surgery with the intraoperative deviation (3). The observer was not masked as to the preoperative awake deviation in each patient, but the observer was masked in a historical sense because the data grouping represented in this study was not devised until several years after this data was recorded.

RESULTS

Table 1, ABOVE lists some characteristics of each group, including the pre-op angle prior to the first surgery for each group, and also enables comparison of the pre-op angle of esotropia in the awake patients of Groups 2, 3, and 5 which were not significantly different in their initial angles of esotropia (one-way analysis of variance p = 0.476).

Table 2, NEXT PAGE, BELOW OR RIGHT lists the exoshift under anesthesia for each group of exoshifts and gives relevant comparisons. Group 2 (undercorrected) was virtually identical to Group 1 (prior to any surgery). Group 3 (orthotropic horizontally after the first surgery, receiving a second surgery for a vertical deviation) was significantly reduced compared to Group 1, but was larger than the exoshifts of Group 4 (superior oblique palsy). The exoshifts of Group 4 (superior oblique palsy) and Group 5 (late consecutive exotropia) were not significantly different.
### Table 2. RESULTS: Comparisons of Exoshift Under Anesthesia Among Groups

<table>
<thead>
<tr>
<th>Group #, Description</th>
<th>Exoshift Under Anesthesia</th>
<th>Unpaired Student’s t Test</th>
<th>Mann-Whitney U Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Prior to any Surgery</td>
<td>41.2 ± 15.0</td>
<td>1 vs 2: p = 0.86</td>
<td>p = 0.69</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 vs 3: p &lt; 0.0001</td>
<td>p = 0.002</td>
</tr>
<tr>
<td>2. Undercorrected after MROU</td>
<td>41.8 ± 13.6</td>
<td>2 vs 1: p = 0.86</td>
<td>p = 0.69</td>
</tr>
<tr>
<td>3. Ortho after MROU</td>
<td>30.1 ± 6.0</td>
<td>3 vs 1: p &lt; 0.0001</td>
<td>p = 0.002</td>
</tr>
<tr>
<td>4. Superior Oblique Palsy</td>
<td>24.0 ± 9.3</td>
<td>4 vs 3: p = 0.019</td>
<td>p = 0.040</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 vs 5: p = 0.50</td>
<td>p = 0.68</td>
</tr>
<tr>
<td>5. Consecutive XT after MROU</td>
<td>26.0 ± 8.3</td>
<td>5 vs 4, p = 0.50</td>
<td>p = 0.68</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 vs 3: p = 0.11</td>
<td>p = 0.067</td>
</tr>
</tbody>
</table>

Note: An unpaired student’s t test was used, because this is a comparison of the distributions of a measurement among different groups. Due to the moderately small group sizes (N), a non-parametric test, the Mann-Whitney U test, was also calculated.

ET = esotropia, XT = exotropia, MROU = bilateral medial rectus recession

*Text continued on next page, overleaf or below*
Table 3, below compares the exoshifts of the first vs. the second surgery within Group 2 (undercorrected after the first surgery), Group 3 (orthotropic after the first surgery), and Group 5 (late consecutive esotropia). Using these within group comparisons, Group 2 (undercorrected) was unchanged, Group 3 (orthotropic horizontally after the first surgery) was significantly reduced, and Group 5 (consecutive esotropia) was significantly reduced.

Reliable conclusions from comparing the exoshift under anesthesia both among these 5 groups and within groups 2, 3, and 5 depend on the assumption that the patients in groups 2, 3, and 5 were essentially similar prior to surgery and were not sorted by the results of the first surgery into groups with different sizes of angles of esotropia before the first surgery. Comparison of the pre-operative angle of esotropia prior to the first surgery among those undercorrected, fully corrected, and overcorrected by the bilateral medial rectus recessions (Groups 2, 3, and 5) was tested with a one-way analysis of variance and no significant difference was revealed (p = 0.476). Table 2 on prior page above details comparison of the exoshift under anesthesia among all 5 groups. Because this is a comparison of the distributions among different groups, an unpaired student's t test was used. Because the group sizes were moderately small, a non-parametric test, the Mann-Whitney U test, was also calculated. The p values calculated by these two different methods lead to identical conclusions in regard to which differences are significant.

Similarly, Table 3, prior page details comparison of the exoshift under anesthesia at the

### Table 3. RESULTS:

**Comparisons of Exoshift Under Anesthesia at First and Second Surgery within Groups**

<table>
<thead>
<tr>
<th>Group #, Description</th>
<th>Exoshift Under Anesthesia at First Surgery</th>
<th>Exoshift Under Anesthesia at Second Surgery</th>
<th>Student’s t, Wilcoxon Ranks test</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Undercorrected after MROU</td>
<td>41.9 ± 13.2</td>
<td>41.8 ± 13.6</td>
<td>p = 0.96 (p = 0.69)</td>
</tr>
<tr>
<td>3. Ortho after MROU</td>
<td>38.9 ± 19.3</td>
<td>30.1 ± 6.0</td>
<td>p = 0.054 (p = 0.041)</td>
</tr>
<tr>
<td>5. Consecutive XT after MROU</td>
<td>42.4 ± 13.1</td>
<td>26.0 ± 8.3</td>
<td>p &lt; 0.001 (p = 0.001)</td>
</tr>
</tbody>
</table>

Note: A paired student’s t test was used, because this is a comparison of a repeated measurement within a group before and after MROU. Due to the small group sizes (N), a non-parametric test, the Wilcoxon signed ranks test, was also calculated, and is the second p value listed for each group.

MROU = bilateral medial rectus recession
start of the first and the second surgery for Groups 2, 3, and 5. Because this is a comparison of repeated measurement within a group before and after bilateral medial rectus recessions, a paired student's t test was calculated. Because of the small group sizes, a non-parametric test, the Wilcoxon signed ranks test, was also calculated, and is the second p value listed for each group. The strong trend in reduction of exoshift under anesthesia at the second surgery among those fully corrected by the bilateral medial rectus recessions (Group 3) demonstrated by the paired student's t test (p = 0.054) is verified as a significant reduction by the Wilcoxon signed ranks test (p = 0.041). The non-parametric test is probably more valid. p values for other comparisons in Table 3 calculated by these two different methods lead to identical conclusions in regard to Groups 2 and 5.

Table 4, below details comparisons of the positions under anesthesia among the 5 different groups in a manner identical to the techniques used in Table 2. In all listed comparisons the differences were significant by both types of statistical analysis.

### Table 4: RESULTS: Comparisons of Position under Anesthesia Among Groups

<table>
<thead>
<tr>
<th>Group #, Description</th>
<th>Position</th>
<th>Unpaired Student's t Test</th>
<th>Mann-Whitney U Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Prior to any Surgery</td>
<td>0.9 ± 13.2 ET</td>
<td>1 vs 2: p &lt; 0.0001</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>2. Undercorrected after MROU</td>
<td>22.0 ± 15.1 XT</td>
<td>2 vs 1: p &lt; 0.0001</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>3. Ortho after MROU</td>
<td>30.4 ± 6.1 XT</td>
<td>3 vs 1: p &lt; 0.0001</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>4. Superior Oblique Palsy</td>
<td>24.1 ± 9.3 XT</td>
<td>4 vs 3: p = 0.017</td>
<td>p = 0.036</td>
</tr>
<tr>
<td>5. Consecutive XT</td>
<td>39.5 ± 8.9</td>
<td>5 vs 4: p &lt; 0.0001</td>
<td>p &lt; 0.0001</td>
</tr>
</tbody>
</table>

Note: An unpaired student's t test was used, because this is a comparison of the distributions of a measurement among different groups. Due to the moderately small group sizes (N), a non-parametric test, the Mann-Whitney U test, was also calculated.

ET = esotropia, XT = exotropia, MROU = bilateral medial rectus recession
Table 5 next page, below or right: considers the probable degree of abnormality of contracture and of innervation of the medial recti and the lateral recti in each of the 5 groups, and compares this with the observed exoshift in anesthesia in each group. Probable innervation of the medial recti is the only perfect match.

**DISCUSSION**

Those familiar with the classic, pioneering work of Apt and Isenberg (6) and Romano (7) might suppose that this study merely replicates their finding of a positive correlation between the size of the preoperative esotropia and the exoshift under anesthesia and a negative correlation between the size of the pre-operative exotropia and the size of the exoshift under anesthesia. (In these previous studies, the largest exoshifts were associated with the largest angles of pre-anesthesia esotropia and the smallest exoshifts were associated with the largest angles of pre-anesthesia exotropia.) Study of our data, however reveals three important comparisons among specific groups of our patients which would not be explainable as simple replication of previous studies of large heterogeneous groups of strabismus patients which included a variety of preoperative clinical circumstances.

First, those whose angle of deviation was only partially reduced by the initial bilateral medial rectus recession (Group 2) had an exoshift under anesthesia virtually identical to the patients with infantile esotropia prior to any surgery (Group 1). (Group 1, N = 60, with a pre-op deviation of 42.1 ± 18.3 ET and exoshift 41.2 ±15.0 vs. Group 2, N = 27, with a pre-op deviation of 19.8 ±7.4 ET and exoshift 41.8 ±13.6.) Thus, among the 27 who experienced a 50% reduction in the size of their esotropia after the initial bilateral medial rectus recession, there was still no reduction at all in the size of the exoshift under anesthesia. It is clear that mechanical factors alone reduced the deviations after undercorrecting surgery (probably increased lateral rectus contracture due to the increased exodeviation while sleeping after bilateral medial rectus recession), and that standard recessions do not change innervational status without successful eye alignment. (It is likely that the position of the eyes while sleeping is similar to the position of the eyes under anesthesia. Note in Table 4 the 22.0 ±15.1 XT position under anesthesia of Group 2, Undercorrected after MROU, highly significantly different from the 0.9 ±13.2 ET position under anesthesia of Group 1, Prior to any Surgery.)

The second result of a comparison among specific groups of patients not explainable as simple replication of previous studies was that the exoshift under anesthesia measured at the second surgery among those made horizontally orthotropic (5ET to 5XT; See reference 8) by the initial bilateral medial rectus recession (Group 3, who were receiving a second surgery for only a vertical deviation, inferior oblique overaction or Dissociated Vertical Deviation) was significantly larger than the exoshift under anesthesia among a group of superior oblique palsy patients who had no horizontal deviations (Group 4). (Group 3, N = 17, with a pre-op deviation of 0.3 ±2.7 XT and exoshift 30.1 ±6.0 vs. Group 4, N = 21, with a pre-op deviation of 0.1 ±2.0 XT and exoshift 24.0 ±9.3, p = 0.019 by unpaired Student's t test and p = 0.040 by Mann-Whitney U test.) Although this difference was relatively small, it was significant. We conclude that the exoshift under anesthesia parallels the level of innervation of the medial recti and that patients made orthotropic by the initial bilateral medial rectus recession still require a small amount of hyperinnervation of the medial rectus muscles to maintain orthotropia. This small amount of hyperinnervation could be "needed" to maintain orthotropia after bilateral medial rectus recession to counter the increased contracture of the lateral rectus muscles and, for the larger bilateral medial rectus recessions, the effect of torque vector reduction (9). Measurement of lateral rectus contracture in these groups could be a fruitful field of future research.

The third important result of a comparison among specific groups of patients not explainable as simple replication of previous studies was that the exoshift under anesthesia measured at the second surgery among those receiving a second surgery due to late consecutive exotropia (Group 5) was virtually identical to that of superior oblique palsy patients with no horizontal deviations (Group 4) in spite of a vast difference in pre-op deviations. (Group 5, N = 16, with a pre-op deviation of 13.5 ±6.8 XT and exoshift 26.0 ±8.3 vs. Group 4, N = 21,
Table 5. RESULTS:

Which of 4 Probable Factors Correlates Most Closely with the Exoshift Under Anesthesia?

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Probable Contracture of Lateral Recti</td>
<td>much less than normal</td>
<td>a little less than normal</td>
<td>a little more than normal</td>
<td>normal</td>
</tr>
<tr>
<td>Probable Innervation of Lateral Recti</td>
<td>much less than normal</td>
<td>much less than normal</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>Probable Contracture of Medial Recti</td>
<td>more than normal</td>
<td>very much more than normal*</td>
<td>much more than normal*</td>
<td>normal</td>
</tr>
<tr>
<td>Probable Innervation of Medial Recti</td>
<td>much more than normal</td>
<td>much more than normal</td>
<td>a little more than normal</td>
<td>normal</td>
</tr>
</tbody>
</table>

Observed Exoshift under Anesthesia | much more than normal | much more than normal | a little more than normal | normal | normal |

Note that the last two lines of this table of probable factors are a perfect match!

* There is very much more contracture than normal in the MR among undercorrected patients (Group 2) and much more contracture than normal among fully corrected patients after MROU than prior to MROU because the mm of recession are about double what would be required for geometric straightening.
with a pre-op deviation of 0.1 ±2.0 XT and exoshift 24.0 ±9.3, p = 0.50 by unpaired Student's t test and p = 0.68 by Mann Whitney U test. We have concluded that the level of innervation of the medial rectus muscles has become normal in those patients with late consecutive exotropia, none of whom were undercorrected or overcorrected at 6 weeks after the initial surgery. Thus, the medial rectus muscles can no longer compensate for the lateral rectus contracture present after previous bilateral medial rectus recession or for the reduction in torque vector after larger bilateral medial rectus recessions. In Table 4 it is demonstrated that the position of the eyes under anesthesia (the right end of the bars in Figure 2, see next page) is more exotropic among those patients in Group 3 (fully corrected) than among those in Group 4 (normal medial rectus innervation) and still more exotropic among those in Group 5 (late consecutive exotropia). The position of the eyes under anesthesia is probably similar to the position of the eyes while the patient is sleeping, and the larger angles of exotropia while sleeping would provide an opportunity for increased contracture of the lateral recti. For Group 3, the abnormally exotropic position is occurring only when the patient is asleep. For Group 5 the abnormally exotropic position is occurring both when the patient is asleep and when the patient is awake. The comparisons detailed in the preceding paragraphs suggest that the medial rectus muscles in Group 3 remain slightly hyperinnervated until and unless the late consecutive exotropia of Group 5 develops and the hyperinnervation normalizes. Since the probable increased contracture of the lateral recti in Group 3 is likely to be due to the geometric repositioning of the eyes during sleep produced by the previous bilateral medial rectus recessions, one important prediction of this line of reasoning is that children with infantile esotropia made orthotropic with botulinum injections would have a normal position of the eyes while under anesthesia or while sleeping, would not have abnormal contracture of the lateral rectus muscles, would not need a small degree of relative hyperinnervation to keep the eyes straight and therefore later in life would have an almost zero incidence of late consecutive exotropia. This prediction has been recently verified by Tejedor and Rodriguez of Madrid, who found only 7 consecutive exotropes among 2,445 such children (10).

Table 5 considers the probable contracture and innervation of the medial and lateral recti in each of the 5 groups. Each of the comparisons with normal in this table is simple common sense, keeping in mind that the position of the eyes while sleeping is probably similar to the position observed in this study under anesthesia. The general principle regarding contracture is that a muscle, over time, reorganizes itself to have a normal linear density of sarcomers according to its average habitual length, regardless of its level of innervation. Two comparisons in Table 5 which deserve special comment are the probable contracture of the medial recti in Group 2 Undercorrected after MROU and the probable contracture of the medial recti in Group 3 Ortho after MROU. There is very much more contracture than normal in the MR among undercorrected patients (Group 2) and much more contracture than normal among fully corrected patients after MROU because the mm of recession are about double what would be required for geometric straightening.

Mechanical orbital forces, excluding the probable contracture (abnormal shortening) and expansure (abnormal lengthening) of the horizontal recti detailed in Table 5, are probably weak and of minimal importance in determining the position of the eyes under anesthesia. If they were of any significance, there would be a correlation between the size of the exoshift under anesthesia and the proximity of the position of the eyes under anesthesia to a specific point of rest where, presumably, the non-muscular forces would be in balance. Casual inspection of Figure 2 reveals no such point.

Finally, we would like to respond to two criticisms made by thoughtful colleagues. First, the suggestion has been made that another control group could be generated from patients who were undergoing other types of eye surgery. All of these data came from the practice of the primary author between 1990 and 1998, and the surgical practice of this author has been limited to strabismus since 1985. Also, consistency of anesthesia technique has undoubtedly been critical to the validity of the comparisons made in this study, and the anesthesiologist for these surgeries is no longer available. This is why we used superior oblique palsy patients as the control group, Group 4.

Second, some of the differences among groups described in this study which we feel contribute to our understanding of how successful bilateral medial rectus recessions work for infantile esotropia are small. There is an essential difference in a basic science study, in which small but significant differences can be very informative (such as described in the biography of Madame Curie by her daughter) (11), and a clinical study, in which small differences my be statistically different but not clinically important. Hopefully, this study is properly regarded as one using clinical data to inform a basic science question.
Figure 2. (Mims III et al) Exoshift under anesthesia among Groups 1 through 5. The left end of each shaded bar corresponds to the angle of esotropia measured in the clinic pre-operatively with the patient awake. The right end of the shaded bar corresponds to the angle measured under anesthesia at the start of the surgery. The length of the bar equals the exoshift under anesthesia.
REFERENCES

8. Mims III JL. The oval of adequate binocular alignment: just how straight so the eyes of an infantile esotrope have to be maintained to preserve stereopsis and binocular vision? Binocular Vis Strabismus Q 2002;17:281-286.
Macular and Retinal Nerve Fibre Layer Thickness in Strabismic and Anisometropic Amblyopia

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ABSTRACT: Electrophysiological studies investigating the relationship between amblyopia and retinal function have in the past produced conflicting findings, leaving the retinal correlates of amblyopia, if they exist, unknown. Recent advances in technology, and in particular the emergence of optical coherence tomography, has resulted in further investigations of the retinal nerve fibre layer and macula of amblyopia patients using these instruments. This review summarises the recent literature in this area and discusses various issues relating to this research.

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INTRODUCTION

Amblyopia has long been known to have a cortical and lateral geniculate basis, however, the involvement of further structures, and in particular the retina, remains questionable. Establishing the presence or absence of retinal involvement in amblyopia is important, as it may have implications in its treatment and prognosis. Early electrophysiological data investigating ocular involvement in amblyopia has been inconclusive. To address this, new optical imaging technology has recently been employed to further investigate the possibility of retinal involvement in amblyopia. These instruments may be able to detect subtle differences in macular and retinal nerve fibre layer thickness in amblyopic eyes, which has previously not been possible. This paper reviews the current literature investigating retinal involvement in amblyopia using new optical imaging technology.

AMBYLOPIA

Amblyopia is a decrease in vision in one or both eyes, occurring in the absence of structural or pathologic anomaly and persisting after the correction of refractive error. It is deemed to be reversible by therapeutic measures and is conventionally divided into four types: anisometropic, strabismic, ametropic and visual deprivation. Abnormal binocular interaction or competition and deprivation of form vision are the causative mechanisms involved in the development of amblyopia.

It has long been known that the amblyopic process has an effect at various levels of the visual pathway; including loss of binocularly driven cells, shifts in ocular dominance in the visual cortex and cell shrinkage in layers of the lateral geniculate nucleus and columns in the visual cortex. Further involvement of ocular structures, the retina in particular, remains controversial. Electrophysiological studies investigating possible retinal involvement are conflicting and inconclusive. Whilst several studies have shown that pattern electroretinograms and electro-oculograms in ambyopic eyes are reduced as compared to normals, other studies have not concurred.

INSTRUMENTATION

Optical coherence tomography (OCT), scanning laser polarimetry (SLP) and confocal scanning laser ophthalmoscopy (CSLO) are the latest technological developments that enable a more reliable, objective and reproducible measurement of retinal features and retinal nerve fibre layer (RNFL) thickness. To date, the major clinical applications of these devices are in the diagnosis of glaucoma and the assessment of retinal pathologies, such as detachments, macular holes and macular oedema. Recently, studies examining retinal involvement in amblyopia have used these new optical imaging instruments to investigate RNFL thickness in strabismic and anisometropic ambylopies. The RNFL arises from the axons of ganglion cells, with the number of axons in the retina decreasing during gestation. It has been suggested that retinal ganglion cells may be involved in the amblyopic process, suffering modifications due to inadequate stimulation, thus affecting postnatal reduction and resulting in an abnormal RNFL thickness.

Most of the recent studies examining retinal involvement in amblyopia have used OCT and SLP technology. While there are differences between the two instruments, both are capable of generating highly accurate and
reproducible RNFL assessments\textsuperscript{12-14} and comparative studies have shown that measurements of RNFL thickness correlate well between the two instruments\textsuperscript{26,27}.

The OCT uses infrared light, which is projected onto the retina, to produce cross-sectional images of the retina by measuring the time delay and intensity of light reflected back from different structures in the eye\textsuperscript{28}. The OCT is capable of measuring both the thickness of the RNFL in the region surrounding the optic nerve, i.e. the peripapillary RNFL and the thickness and volume of the macular region\textsuperscript{28}. The SLP, on the other hand, provides RNFL thickness measurements based upon the birefringence of microtubules in the retinal ganglion cell axons\textsuperscript{29}. Birefringence occurs when polarized light passes through a birefringent medium, such as the RNFL, causing it to split into two waves, with each component travelling at a different speed. This creates a phase shift called retardation, with the amount of retardation being proportional to the thickness of the RNFL\textsuperscript{29}. The SLP measures only the thickness of the peripapillary RNFL, in contrast to the OCT, which is also capable of measuring the thickness of the macular region.

CURRENT STUDIES OF MACULAR THICKNESS IN AMBLYOPIA

Most OCT studies investigating macular thickness in amblyopes have not reported a significant difference in thickness between amblyopic and fellow normal eyes\textsuperscript{15,\textsuperscript{20,24}}. A small study by Kowal and Wong\textsuperscript{30}, however, compared the macular thickness of successfully and unsuccessfully treated amblyopia patients, and reported a significantly thicker macular to exist only in those patients where amblyopia treatment was ineffective in improving visual acuity. They suggested that the OCT may therefore be able to detect subtle changes in the macular of amblyopic eyes and hence be an indicator of prognosis during treatment. However, macular thickness values may have been falsely increased due to inadvertent measurement of a parafoveal eccentric point in at least some anisometric amblyopes.

It is known that a high number of anisometric amblyopes display a parafoveal eccentric fixation related to their amblyopia. This ‘microtropia with identity’-like phenomenon presents with a small-angle strabismus and an associated eccentric fixation ‘point’, more or less equal to the angle of the deviation\textsuperscript{31}. This adaptation means that the amblyopic eye will display parafoveal fixation under both monocular and binocular viewing conditions. A study by Hardman Lea et al\textsuperscript{31} showed that anisometropic amblyopia is a common feature of microtropia (and vice versa), with an estimated 45\% of the anisometropic amblyopes in their series of patients displaying parafoveal fixation. When measuring macular thickness with the OCT, the patient fixates on a target and the light beam is aimed at the fixing retinal structure. However, the OCT is unable to detect whether a patient is actually fixing foveally. Given that macular thickness increases parafoveally, measuring a slightly eccentric fixation point presumed to be the foveal pit (or the ‘umbo’) will cause macular thickness values to be falsely increased\textsuperscript{32}. This limitation of the OCT was first pointed out by Wilkinson\textsuperscript{32} in a critical appraisal of recent studies’ reports of increased retinal nerve fibre layer thickness in amblyopic eyes relative to fellow normal eyes.

Hence, studies wherein macular thickness was investigated and the presence of eccentric fixation not evaluated in
anisometropic participants, are essentially flawed. It is therefore impossible to conclude whether a true macular anomaly exists in this series of patients. This is an important consideration for future studies undertaking macular thickness investigations in anisometropic amblyopes.

In contrast to measurements of macular thickness, measurements of peripapillary RNFL thickness are not affected by eccentric fixation. When measuring RNFL thickness, the patient fixates on a target and the examiner positions a target circle at the optic nerve head. Thus, whether a patient is fixating foveally or parafoveally will not affect thickness values.

CURRENT STUDIES OF RNFL THICKNESS IN AMBLYOPIA

Like studies using new technologies to investigate macular thickness in amblyopes, those investigating RNFL thickness in amblyopes remain inconsistent. Whilst several studies using SLP technology have found no significant differences in RNFL thickness between amblyopic and fellow normal eyes in either strabismic or anisometropic amblyopia, OCT studies are divided. Kee et al reported no significant difference in RNFL thickness between amblyopic and normal eyes. However, Yen et al found that refractive (<2D difference in spherical equivalence between the two eyes) and anisometropic (≥2D difference in spherical equivalence) amblyopes had a significantly thicker peripapillary RNFL when compared to fellow non-amblyopic eyes. This difference, however, was not found in their series of participants with strabismic amblyopia. The authors of this study suggested that the process of postnatal reduction of ganglion cells may therefore require sharply focussed objects as stimuli. This finding is further supported by OCT studies which report no significant difference in peripapillary RNFL thickness between the amblyopic and fellow non-affected eyes of individuals with strabismic amblyopia and by Yoon et al who reported a significantly thicker peripapillary RNFL in their series of participants with anisohypermetropic amblyopia.

STRABISMIC VERSUS ANISOMETROPIC AMBLYOPIA

Psychophysical studies have shown that strabismic and anisometropic amblyopia represent two separate and distinct disorders. For instance, whilst strabismic amblyopia demonstrates reduced spatial frequencies for particular gratings with decreasing luminance as well as severe loss in acuity and ‘crowding effects’ with vernier gratings, anisometropic amblyopia does not exhibit these defects. Evidence of these different visual characteristics suggests that different neural losses are associated with each type of amblyopia. OCT studies have also recently found that a significantly thicker RNFL exists in anisometropic and refractive amblyopic eyes in comparison to fellow non-amblyopic eyes and that this is not the case in strabismic amblyopic eyes. Kee et al also reported a significant difference in thickness between anisometropic and strabismic amblyopes. This was such that the macular was found to be thicker in strabismic amblyopes (as in other studies), the RNFL was thicker in anisometropic amblyopes. This suggests that there may be ‘retinal characteristics’ that differ between the two disorders.
However, it should be noted that whilst Yen et al\textsuperscript{23} found a significantly thicker RNFL in patients with refractive and anisometropic amblyopia (relative to the fellow normal eye), they failed to accurately divide their participants into types of amblyopia as appropriate. They initially divided their series of 38 patients with unilateral amblyopia into either a refractive or strabismic group. Of these, 19 were also classified as having anisometropic amblyopia (defined as a difference in spherical equivalence of ≥2D), 12 being from the refractive group and 7 from the strabismic group. Thus, the anisometropic group included participants that were also defined as having only strabismic amblyopia. As such, the results of Yen et al\textsuperscript{23} were confounded by the unclear delineation between types of amblyopia. The results of studies comparing the RNFL of different types of amblyopia are further confounded by the finding that RNFL thickness measurements may correlate with axial length or refractive error\textsuperscript{36-38}. In brief, and as discussed in the following section, any differences found between strabismic and anisometropic amblyopes may only be related to the presence of refractive error and not the aetiology and the pathophysiology of the amblyopia.

**REFRACTIVE ERROR**

Although current research has suggested that retinal ganglion cells in anisometropic amblyopia may be involved in the amblyopic process, studies using the OCT have shown that RNFL thickness measurements are influenced by refractive error alone\textsuperscript{38}. Salchow et al\textsuperscript{38} found that refraction of the eye is correlated with RNFL thickness, such that myopes have a thinner RNFL and hypermetropes a thicker RNFL in comparison to emmetropes. Similar findings were reported in a large study conducted by Huynh et al\textsuperscript{39} investigating RNFL thickness in a normal population of six year old children.

Given that the majority of the anisometropic amblyopes included in the studies by Yen et al\textsuperscript{23} and Yoon et al\textsuperscript{24} were hypermetropic, the significantly thicker RNFL found in anisometropic amblyopes as compared to normals may not be directly related to amblyopia, rather to refractive error. Salchow et al\textsuperscript{38} found that for each dioptre of hypermetropia, RNFL thickness increased by approximately 1.7 microns. Therefore, the only way to attribute the increased thickness in anisometropic participants to amblyopia is to compare non-amblyopic hypermetropes to a comparable group of hypermetropes with amblyopia. As neither study ‘controlled’ for this, it is impossible to determine whether the increased RNFL thickness was due to amblyopia or an organic characteristic of the eye. Whilst Yen et al, did include a control group, this consisted of mainly myopic participants. The RNFL thickness in myopes is, however, contentious with several studies suggesting that myopes do not have a thinner RNFL\textsuperscript{40-43} in comparison to emmetropes, and others reporting that increased axial length correlates with a thinner RNFL\textsuperscript{36, 37}.

The influence of refractive error on RNFL thickness has also been studied by Atilla et al\textsuperscript{16} using SLP and the Heidelberg Retinal Tomography (HRT) (a CSLO instrument). In this study, similarities between hypermetropic and anisohypermetropic amblyopic participants were found in comparison to emmetropic participants. The hypermetropic and anisohypermetropic amblyopic groups consisted of participants with similar refractive errors. No significant
differences were found between either group in the various parameters taken with the SLP. The HRT, on the other hand, revealed a significantly smaller disc area and cup/disc (C/D) ratio in the hypermetropic and anisohypermetropic amblyopic participants in comparison to the emmetropic group. No such significant difference was found between the hypermetropic and anisohypermetropic amblyopic groups, however. This lends further support to the theory that amblyopia does not have an ocular anatomic site and that RNFL pathologies found in anisometropic patients using OCT studies may be an artefact of refractive error.

RNFL THICKNESS AND VISUAL ACUITY IN AMBLYOPIA

Although there is little data on the use of the OCT, SLP and CSLO in children, their use in adults has been widely investigated, particularly in glaucoma. Results of these studies have shown good correlation between the OCT and SLP with visual field defects, suggesting they may also be a good predictor of functional vision. Furthermore, studies have shown that macular and RNFL thickness may be associated with visual acuity, such that a thinner or thicker RNFL to the norm are associated with decreased visual acuity.

There is currently little data available, however, correlating RNFL measurements in amblyopia with visual acuity. Yen et al. and Kowal and Wong are the few studies, to date, to have investigated this relationship between RNFL and visual acuity in amblyopes. Yen et al. found that RNFL thickness in amblyopic eyes did not correlate with visual acuity after adjustment for age. This is in contrast to Kowal and Wong, however, who reported that patients with no improvement in visual acuity had a significantly thicker macular in comparison to those whose vision improved following appropriate amblyopia treatment. This suggests that treatment efficacy and decreased visual acuity in amblyopia may be correlated with macular thickness. However, as discussed previously, Kowal and Wong did not account for the possibility that some anisometropes may have had eccentric fixation, thus, falsely indicating a greater macular thickness measurement. As such, it is unclear whether an amblyopic defect was responsible for the increased thickness and hence decreased visual acuity. It is also important to note that this was a small study (involving 12 participants) lacking statistical power. Larger studies which account for the presence or absence of eccentric fixation are needed before it may be determined whether macular or RNFL measurements may be used as a prognostic indicator in the treatment of amblyopia.

CONCLUSION

As with past electrophysiological studies, current research using optical imaging technology to investigate retinal involvement in the amblyopic process has reported conflicting findings. The results in these studies have been particularly confounded by the presence of refractive error and possibly the presence of eccentric fixation. In order to address these issues, future studies need to include appropriate control groups and assess patients’ fixation pattern. Furthermore, optical imaging technology must progress whereby it has the ability to determine and measure the umbo rather than the fixation point. It is important to continue investigating correlations between retinal thickness and amblyopia, as this relationship could influence the prognosis of amblyopia treatment and final visual acuity outcomes.
REFERENCES


Case Report

Unilateral Myelinated Nerve Fibers Associated with Hypertropia, Strabismus and Amblyopia. ? Reverse Straatsma Syndrome ?

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INTRODUCTION

One of many “strabismus syndromes” is that of strabismus with myelinated retinal nerve fibers associated with ipsilateral myopia, amblyopia, and strabismus. It has been well described in the literature since Straatsma and associates first reported this association in 1979 when they described four patients with the above features who all had profound visual impairment.

Different mechanisms have been proposed to account for this association, including myelinated nerve fibers causing myopia, or myopia leading to myelination of the nerve fibers. To our knowledge, in all of the reported cases, the eyes with myelinated nerve fibers were either more myopic or less hyperopic compared to the uninvolved eye. In this article we report one case of unilateral myelinated nerve fibers associated with hyperopic anisometropia and amblyopia. We propose to name it reverse Straatsma syndrome.

CASE REPORT

A 2.5 year old girl from Columbia was referred for amblyopia evaluation. Before moving to the United States, the patient was seen by an ophthalmologist in Columbia who diagnosed her with myelinated nerve fibers OS. She was offered glasses of +1.00 sphere OD and +4.50 sphere OS and instructed to patch OD although patient and family reported poor compliance. On our exam, both her eyes fixed and followed with a microstrabismus of 4 prism diopters of esotropia. Pupils were equal, round, and reactive to light and accommodation, with no afferent pupillary defect. Cycloplegic refraction revealed OD: +2.50-0.75x40 and OS: +6.00-1.25x135. Fundus examination demonstrated peripapillary myelinated nerve fibers OS, (see Figure, below) while the right fundus was
unremarkable. Patient was offered new glasses and instructed to patch her right eye 4 hours a day. After 7 months of therapy, patient’s visual acuity was documented for the first time, which was 20/30 OD and 20/40 OS. At 3 years and 11 months of age, her visual acuity improved to 20/25 OD and 20/30 OS. The cycloplegic refraction at this time was OD: +1.25-0.50x45 and OS: +4.50-2.50x145.

DISCUSSION:

Since Straatsma and associates\(^1,2\) described the syndrome of myelinated nerve fibers associated with ipsilateral myopia and amblyopia, there have been many articles reporting this syndrome, discussing the visual prognosis\(^6\), and trying to explain the association of these features\(^3,4,7\). There have been conflicting reports regarding the pathogenesis as well as the visual prognosis. Schmidt and associates\(^1\) reported 13 patients with unilateral myelinated nerve fibers divided into two groups based on the location of myelination. They found that all six patients with wide-spread myelination extending to the midperiphery of the retina also had strabismus, severe amblyopia, and unilateral myopia. On the other hand, all seven patients with circumscribed myelinated nerve fibers localized merely around the optic disc had near-normal visual acuity, no distinct refractive error, and no strabismus. They concluded that myopia only occurred in eyes with wide-spread myelinated nerve fibers but not in eyes with circumscribed myelinated nerve fibers. Furthermore, they made the assumption that wide-spread myelinated nerve fibers cause myopia by inducing blurred retinal image, in analogy to the development of myopia in congenital opacification of the optical system. However, it is questionable how much visual deprivation can the myelinated nerve fibers really cause, especially since they only lead to partial scotomas in the periphery, sparing the fovea. Not only are those partial scotomas generally smaller that predicted by the extent of myelination\(^2\), their effect on refractive errors is also debatable. The results of recent experiments in animals trying to address the question whether peripheral defocus affect central refractive development have been inconsistent in monkey and chicken so far.\(^8,9\)

Lee and Gonzalez\(^4\) proposed that peripapillary myelinated nerve fibers in a unilateral myopic eye may be secondary to an imbalance between the process of myelination and the formation of the lamina cribrosa. A myopia eye may be predisposed to this imbalance because it has a greater axial length. Doubt has been casted on this theory as well because it cannot explain myelinations discontinuous with the optic nerve head.\(^10\) However, it is likely that congenital myelinated nerve fibers occur in a complicated developmental process that multiple risk factors are involved and myopia is just one of them.

To our knowledge, there has not been described in the literature that unilateral myelinated nerve fibers are associated with hyperopic anisometropia and amblyopia. Ruttum and Poll\(^10\) reported an interesting variation of the syndrome in which they described two cases with amblyopia developing in the contralateral eye. One patient had a refractive error of -2.50 in the eye with myelinated nerve fibers and +7.50 in the other eye which had amblyopia. The other patient had a refractive error of +1.00 in the eye with myelinated nerve fibers and +2.75+1.50x90 in the eye with amblyopia. They suggested that these two patients are genetically predisposed to be hyperopic, and because of the relative elongation of the eye with myelinated nerve fibers, their involved eyes became relatively emmetropic and amblyopia developed in the contralateral eyes. Although one of their patients was hyperopic, the involved eye was still axially more elongated compared to the uninvolved eye.

One of the patients reported by Schmidt and associates\(^1\) also had a refractive error of +5.0-1.0x100 in the involved eye and +6.0-1.0x90 in the uninvolved eye. However, this patient was only listed in their table as patient 7 who is a 72-year-old woman with visual acuity of 20/30 in the involved eye and 20/40 in the uninvolved eye. No detailed case description was available. We do not know whether her myelinated nerve fibers were present at birth or acquired later in life. We do not know whether she had cataracts or other concomitant conditions to explain the worse visual acuity in the uninvolved eye. But apparently, she did not have amblyopia. The bottom line is, she did not have significant anisometropia and her involved eye was still less hyperopic than the uninvolved eye.

In contrast, our patient’s involved eye is significantly more hyperopic with 3.25 D of anisometropia on presentation that slowly reduced to
2.25 D after 16 months of therapy. The association of myelinated nerve fibers with hyperopic anisometropia and amblyopia argues against the theory that myelinated nerve fibers cause myopia. Kee and Hwang indicated in their study that even with widespread myelinated nerve fibers, only 9 out of 12 patients had a unilateral myopia of -5.75 D or more and only 6 out of 12 patients had strabismus. However, they did point out that area of myelination, measured as clock hours, together with the amount of anisometropia, is the two most important prognostic factors in this syndrome. We believe that patients with widespread myelination as suggested by Schmidt and associates, or larger area of myelination as mentioned by Kee and Hwang, do represent a separate entity from patients with smaller area of myelination and/or localized around the optic disc. The former is more likely to have high myopia, amblyopia, strabismus, and poor visual outcome. They represent the classic syndrome described by Straatsma and associates. The latter on the other hand, has non-predictable refractive error and may be associated with other eye conditions including amblyopia and strabismus by random chance.

We believe that is the case for our patient whose myelinated nerve fibers are rather localized to the optic disc area. It is likely a coincidence that she has hyperopic anisometropia which is not an uncommon condition in children. We do not know why there have not been more reports on this association if these two features could independently coexist by chance. However, until more cases are published, there is no evidence to suggest a cause and effect relationship between unilateral myelinated nerve fibers and hyperopia.

Although successful treatments with good visual outcome have been reported, most cases in the literature were refractory to standard amblyopia treatment. Macular appearance, amount of anisometropia, and area of myelination have all been suggested as prognostic factors. Our patient achieved an excellent response to amblyopia therapy, further suggesting that this case with rather localized myelinated nerve fibers probably represents a separate entity from the classic syndrome described in the literature. Therefore we propose to name it reverse Straatsma syndrome.

REFERENCES


Followup on a Contributor- Oliver Sacks, M.D. Shares A Binocular Vision Disability with Both a Subject of His Writings Previously Reported Here, and Your Editor = “Poetic INjustice!”

We were surprised to get a followup on one of BV&S Q’s recent guest contributors in a financial newspaper, but it seems he is a New Yorker of some reknown! -per

from The Wall Street Journal September 24, 2008 “A Cultural Conversation with Oliver Sacks” by Judith H. Dobrzynski. A Still Restless Mind at Age 75. “You may think of Oliver Sacks, the renowned teller of weird but true tales about the vagaries of the human brain, as a neurologist first and an author second. If he had to choose, he would disagree. He aspired to be a writer at age 12, a feat he accomplished decades ago, with the publication of “Migraine” in 1970 and “Awakenings” in 1973. At 75, with his 10 books, he has achieved far more: His clinical but compassionate yarns have changead the nature of medical writing. Pressed a little, Dr. Sacks concurs: ‘For better or worse, or both, it’s true that I’ve played a part in putting careful narrative back in medical writing’. When he started, case write-ups emphasized diagnosis, not story. … Dr. Sacks drew inspiration for Alexander Luria’s famous 19th century case history of a man with a limitless memory. There’s also a journal, Neurocase, that published case histories as narrative. And Dr. Sacks’ stories have become movies, plays, operas and TV shows. ‘Human beings are storytellers, and I am a story teller,’ he explains, recalling that he came to the U.S. from Britain in 1960 with an itch to write but unsure of what to write about and whether he should stay in medicine. Only after he learned the enthralling stories of his migraine patients did he find the plot line of his life… …

[One of his stories was published in these pages. “Stereo Sue: Regaining binocular stereoscopic vision in adulthood. A case report. A Neurologists notebook. Stereo Sue. Why two eyes are better than one.” BV&SQ 2006; 21:160 This periodical attracted Dr. Sacks attention by virtue of a series of articles in which your Editor described his difficulties losing his binocularity and stereopsis for a period after a retinal bleed. Now Sacks has suffered a similar personal experience. And your editor used to visit The Cloisters too, many years ago, when he resided in the New York area but way long before he suffered his binocularity problems…]

“…About two years ago, Dr. Sacks was found to have a tumor in his right eye. Lasering caused him to lose central, but not peripheral, vision in that eye. He can see pretty well with his left eye, but he has lost the perception of depth. So now he has become his own experiment in accommodation, using bouncing balls and other devices to teach himself to make better use of his good eye. It’s working, to a point, he says, revealing an incident he had playing ping pong last summer. ‘I swung with great velocity, and thought I had it, he says with a smile. ‘But … it turned out that my swing missed by four feet.’ So, he concludes, ‘My world is now very flat. Now I go the the Cloisters’ -the Metropolitan Museum’s medieval art branch in upper Manhattan - ‘because I have an affinity for 13th century paintings’ made before artists learned to paint with perspective. But Dr. Sacks is sure that ‘learning remains available throughout life, though it’s some what slower as one get old’. He adds: ‘This old brain can still take on a big subject, and I think I will continue to do so unless I get Alzheimer’s. I had an MRI recently, and it looked like the brain of a 30 year old.’” (Ms. Dobrzynski, based in New York writes frequently about the arts and philanthropy.)

Meeting Announcement

In 2009, Asia-ARVO will take place in India where it is being organized by the Indian Eye Research Group. The meeting will be held at the Hyderabad International Convention Center, Hyderabad from January 15-18, 2009. Asia-ARVO is an international meeting developed in cooperation with ARVO to recognize expanding eye research programs in Asia. For information on participation in an AAPOS organized symposium “Hot topics in Pediatric Ophthalmology” contact Michael Repka, MD at mrepka@jhmi.edu/
Istanbul, Turkey. September 22-25, 2010. ISA meeting. This meeting will focus only on strabismus and provides the best opportunity to meet and discuss the topics of most interest to us. Contact: Derek Sprunger, MD at isa.lms@juno.com or visit the website at www.isa.home.org.

**Research Funding Available**

The Blind Children’s Center has funding available to support one year seed grants for research to gain better understanding of visual impairment in children from birth to seven years of age. Grants up to $20,000 will be awarded. Contact: Midge Horton, Executive Director, 323-664-2153 ext 328.

**Number of JCAPHO Certificants Reaches Milestone.**

The Joint Commission on Allied Health Personnel in Ophthalmology (JCAHPO®) announced the number of personnel currently certified by the organization recently exceeded 17,000. Other recent statistics have shown increased enrollment across all levels of JCAHPO certification. The organization currently offers seven levels of certification including three sub-specialty designations. JCAHPO currently has certificants in 19 countries worldwide. For more information on JCAHPO certification or ophthalmic continuing education opportunities visit www.jcahpo.org.

**In Memoriam Ruxandra Sireteanu**

It is never good news to hear that we have lost a fellow worker, especially since there are not that many of us who have devoted our lives to our interest in vision and binocular vision and their pathologies.

We never got to know Ruxandra very well. In fact, I don’t think we ever met her. She did help us out with review of scientific papers at least once.

“By their works shall ye know them” is an ancient truism. And in her memory, on the next two pages we publish her PubMed citations for the last decade. -per
## In Memoriam: RUXANDRA SIRETEANU

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<td>[Spatial misperceptions in amblyopic vision: abnormal activation of the primary visual cortex?]</td>
<td>Sireteanu R, Bäumer C, Sârbu C, Tsujimura S, Muckli L.</td>
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<td>6</td>
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<td>Sireteanu R, Bäumer CC, Sârbu C, Ifthime A.</td>
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In Memoriam: RUXANDRA SIRETEANU

Temporal instability in the perception of strabismic amblyopia.

Cerebral correlates of impaired grating perception in individual, psychophysically assessed human amblyopes.

Children with developmental dyslexia show a left visual "minineglect".

Saliency and context play a role in infants' texture segmentation.

Deficits of spatial localization in children with strabismic amblyopia.

Perceptual learning of highly demanding visual search tasks.

Preliminary report: monocular spatial localization in children with strabismic amblyopia.

Perceptual learning in visual search generalizes over tasks, locations, and eyes.

The binocular visual system in amblyopia.

Live vs. video observation in forced-choice preferential looking: a comparison of methods.

Switching on the infant brain.
Important Warnings


Editorial Note: In an effort to improve efficiency, effective with this issue, we will be editing and further shortening the length of abstracts published here. “Conclusions” will always be preserved and retained but other regular abstract elements will be deleted in so far as they do not significantly (i.e., NOT “statistically p<.05” but simply in our opinion!) influence the conclusions presented. Such abstract details are readily available to all readers on the Internet at PubMed.

For important publications which are not abstracted in PubMed, such as Editorials and Correspondence, we will make efforts to provide our readers with the essence of these items. AUTHORS, PLEASE DO SUBMIT TO US BRIEF ABSTRACTS OF YOUR IMPORTANT EDITORIALS AND PUBLISHED LETTERS TO THE EDITOR regarding strabology and pediatric ophthalmology via email to perxbvq@colorado,net or to judyatbv@vail.net

Vision / Visual Acuity / Amblyopia


Ophthalmology 2008; 115:1796-1799. [Authors Conclusions]

Fixation preference testing, when used as part of a population based research project, does not identify accurately preschool children with 2 lines or more of interocular difference in presenting visual acuity. The clinical value of this test is poor and its use for diagnosis and monitoring interventions should be reconsidered. (Dr. Friedman, Dana Center for Prevention Ophthalmology, Wilmer Eye Institute, Johns Hopkins School of Medicine, Baltimore, Maryland)


We assess four common claims concerning the two-interval forced choice (2-IFC) procedure and the standard Difference Model of 2-IFC performance. The first two are (1) that it is unbiased and (2) that the structure of the 2-IFC task does not in itself alter sensitivity. The remaining two concern a claimed +2 enhancement in sensitivity in 20-IFC relative to that measured in a Yes-No task. We review relevant past research and re-analyze seventeen experiments from previous studies across three laboratories. We then report an experiment comparing 2-IFC performance with performance in a second task designed to elucidate observers’ decision processes. This second task is simply two successive Yes-No signal detection tasks with the same timing as in the 2-IFC experiment. We find little evidence supporting the claims that 2-IFC is unbiased and that it does not alter sensitivity and we also reject the two claims associated with the Difference Model as a model of performance in our own experiment. (Dr. Yeshurun, Dept Psychology, University of Haifa, 31905 Haifa, Israel)


Decreased VA in both eyes of children 30 through 71 months of age at presentation in urban Baltimore was 1.2% among white children and 1.8% among black children. After retesting within 60 days of the initial examination and with children wearing
best refractive correction, the rate of decreased VA in both eyes was 0.5% among white children and 1.1% among black children. (Dr. Friedman, Dana Center for Prevention Ophthalmology, Wilmer Eye Institute, Johns Hopkins Hospital School of Medicine, Baltimore MD)


Conclusions: Among Hispanic/Latino and African American children in Los Angeles County, strabismus prevalence increases with age, but amblyopia prevalence appears stable by 3 years of age. Amblyopia is usually caused by abnormal refractive error. These findings may help to optimize the timing and modality of preschool vision screening programs. (No corresponding author or address given)

A Randomized Trial of Near Versus Distance Activities While Patching for Amblyopia in Children Aged 3 to Less Than 7 Years. Pediatric Eye Disease Investigator Group. Ophthalmology, 2008, in press. [Authors Conclusions]

Performing common near activities does not improve visual acuity outcome when treating anisometropic, strabismic or combined amblyopia with 2 hours of daily patching. Children with severe amblyopia may respond to 2 hours of daily patching.

from the AAOs Academy Express August 27, 2008. Most of the Benefit of Patching and Atropine Persist through Age 10.

The Pediatric Eye Disease Investigator Group previously found that patching and atropine were similarly effective in patients with moderate amblyopia (visual acuity, 20/40-20/100). Investigators in this study evaluated these children at age 10. While improvement in the amblyopic eye is maintained, about half of children initially treated between ages 3 and 7 have mild residual amblyopia at age 10 (visual acuity less than 20/25). Outcome was slightly better in patients treated between the ages of 3 and 5, compared to those treated between ages 5 and 7. (Arch Ophthalmol, August 2008)

Monocular Oral Reading Performance After Amblyopia Treatment in Children. Repka MX,


The monocular oral reading ability when measured with the GORT-4 was slightly worse when reading with previously treated amblyopic eyes compared with fellow eyes in terms of rate accuracy and fluency, but reading comprehension testing was similar. (Dr. Repka, mrepka@jhmi.edu.

Ametropia, Preschoolers’ Cognitive Abilities and Effects of Spectacle Correction. Arch Ophthalmol 2008; 126:252-258. [Authors Conclusions]

Preschoolers with uncorrected ametropia had significant reduction in visual-motor function. Wearing spectacles for 6 weeks improved Beery-Buktenica Developmental Test of Visual-Motor Integration scores to emmetropic levels. (No corresponding address)

Binocular Vision

Local Binocular Fusion is Involved in Global Binocular Rivalry. Takase S, Yukumatsu S, Bingushi K. Vision Research 2008; 48:1798-1803 [Authors Abstract]

We examined whether interocular inhibition in binocular rivalry could occur at the interocular intersection of horizontal and vertical rectangular patches which are locally fusible but globally rivalrous between the two eyes. We measured contrast increment (and decrement) thresholds of a monocularly presented probe which was presented on the horizontal patch corresponding to the intersection. We found that the threshold was higher which the horizontal patch was perceptually suppressed than when it was dominant. In addition, threshold elevation did not occur when both patches were dominant, or when the horizontal patch was viewed in isolation. These results indicate that interocular inhibition occurs at the potentially fusible region, and the determination of binocular fusion or binocular rivalry does not depend on physical stimulus but rather perceptual state at the time. (Dr. Takase, Dept Psychology, Chukyo University, 101-2 Yagotohonmachi, Showa-ku-, Nagoya, Aichi 466-8666, Japan)

There are currently two competing dichotomies used to describe how local stereoscopic information is processed by the human visual system. The first is in terms of the type of the spatial filtering operations used to extract relevant image features prior to stereoscopic analysis (i.e., 1st vs 2nd order stereo). The second is in terms of the temporal properties of the mechanisms used to process stereoscopic information (i.e., sustained vs transient stereo). Here we compare the dynamics of 1st and 2nd order stereopsis using several types of stimuli and find a clear dissociation in which 1st order stimuli exhibit sustained properties while 2nd order patterns show more transient properties. Our results and analyses unify and simplify two complimentary bodies of work. (Dr. Robert Hess, McGill Vision Research, Dept Ophthalmology, McGill University, 687 Pine Ave W (H4-14), Montreal, Quebec, Canada H3A 1A1)

Binocular Ocular Motility


Saccadic eye movements can be used to evaluate different aspects of brain function, and in this article we are concerned with possible applications in relation to anti-epileptic drug treatment. Recent improvements in the technology of measurement have improved the sensitivity and objectivity of the measures. Here we review the neurophysiology of saccades, their classification, their anatomical basis and cortical control, and then published research articles concerned with the influence of anti-epileptic drugs on saccades and their parameters. It seems likely that certain anti-epileptic drugs (especially those acting on ion channels) exert their effect on saccades through ion channels, and this may have relevance to clinical and pharmacogenetic studies. (Dr. Chiening Lo, Inst Neurology, University College London, Queen Square WC1N 3BG, UK. Fax: 44-207-676-2155)


Vertical fusion capability usually increases with convergence. This increase is caused primarily by an increase in the motor component. There is a gradual but small increase in the sensory component as target disparity slowly increases. (Dr. Zee, Johns Hopkins Hospital, 600 North Wolfe St, Baltimore MD 21287-6921)

Strabismus, Pathophysiology


In SO muscle palsy, the contralateral IR muscle is larger and more contractile than the ipsilateral IR muscle, reflecting likely neurally mediated changes that augment the relatively small hypertropia resulting from SO muscle weakness alone. Recession of the hyperfunctioning contralateral IR muscle recession in SO muscle palsy is a physiologic therapy (Dr. Demer, jld@ucla.edu) from the AAOs Academy Express September 25, 2008. CT Scans Fail to Detect Soft Tissue Entrapment in Orbital Floor Fractures in Children About Half the Time.

Researchers retrospectively reviewed all patients (24 pediatric, 31 adults) presenting at a single site for primary repair of orbital floor fracture. They found that pediatric orbital floor fractures are often of the trapdoor type, which require earlier surgical intervention. Radiologists failed to detect entrapment on CT scan in more than 50% of children. Conversely, there was a good concordance between radiologic and intraoperative findings in the adult group. (Ophthalmology, September 2008)

Hypotropic Dissociated Vertical Deviation: A Unique Form of Dissociated Strabismus Complex. Lim HT. Am J Ophthalmol 2008; in press. [Authors Conclusions]

Hypotropic DVD is mostly unilateral and commonly is associated with monocular visual deficits or high myopia. Although the nature of the intermittent slow downward ocular deviation is similar to that of hyper tropic DVD, it should be considered to be a unique form of the dissociated strabismus complex. This rare condition can be corrected surgically by a large recession or a combined recession-resection of the inferior rectus muscle. (Dr. Lim. Htlim@amc.seoul.kr)
Strabismus Pathology
from the AAOs Academy Express August 28, 2008.

Study Finds Diplopia Following Cataract Surgery Occurs in 3 Percent of Patients. This retrospective analysis of patients presenting with diplopia after cataract surgery at a single site during a 70 month period finds a diplopia incidence rate of 3%. Pre-existing strabismus (34%) and extraocular muscle restriction/paresis (25%) were the most common causes of diplopia. Extraocular muscle restriction/paresis was the most common cause of diplopia when infiltration anesthesia was used; pre-existing strabismus was the most common cause with topical anesthesia. (Eye, August 2008)

Strabismus, Surgery


The SO suture spacer procedure alleviated abnormal head positions in patients with Brown Syndrome by improving vertical strabismus in primary position and in the affected field of gaze while avoiding overcorrection in contralateral gaze. The benefits of the procedure persisted over time (Dr. Suh dowsuh@gmail.com)


Without careful search of the Tenon capsule, the condition can be misdiagnosed as an absent tendon. Strengthening the superior oblique tendon in the Tenon capsule can improve the alignment significantly. (Dr. Sato, mihosato@hama-med.ac.jp)

Strabismus Non-Surgical Treatment
from the AAOs Academy Express October 16, 2008.

Office-Based Therapy May Be More Effective that At-Home Treatment for Convergence Insufficiency. Researchers randomized 221 children, ages 9 to 17, to one of three treatment groups: the standard therapy of home-based pencil pushups, home-based computer vergence/accommodative therapy and pencil pushups, office-based vergence/accommodative therapy with home reinforcements, or office-based placebo therapy with home reinforcement. At 12 weeks, nearly 75 percent of children who were given the office-based therapy along with at-home reinforcement achieved normal vision or had significantly fewer symptoms of CI. Only 43% of patients who completed home-based therapy alone showed similar results, as did 33% of patients who used home-based pencil pushups plus computer therapy and 35% of patients given a placebo office-based therapy. (Arch Ophthalmol, October 2008)

Vision Training
from the AAOs Academy Express, April 2, 2008.

Visual Training Can Accelerate Neuroadaptation after Multifocal IOL Implantation. This prospective study included 16 patients implanted with bilateral multifocal IOLs after phacoemulsification. At six weeks postop, they received computer-based visual training for two weeks in one eye. A significant and sustained mean improvement in orientation visual acuity was observed in trained eyes, compared to controls. Contrast sensitivity and near vision under different contrast levels also showed a significant improvement. (J Refractive Surgery, March 2008)


Visual performance after multifocal IOL implantation can be significantly accelerated by a specific 2 week training program. This effect is sustained over a 6 month period. (Ulrich Mester, MD, Dept Ophthalmology., Knappschafts Hospital, D-Sulzbach, Germany. Fax: 49-6897-574-2139. Email: sek-augen@kksulzbach.de)

Dyslexia


In this study, we show that invisible flicker adaptation reduces the perceived duration of a subsequently viewed stimulus in control subjects, but not in dyslexics. Dyslexics, like controls, show
apparent duration compression after 20 Hz flicker and show normal shifts in apparent temporal frequency after adaptation. However, a subgroup of the test group, scoring low on both a test of phonological skill (spoonerisms) and a test of literacy (NART), show an apparent temporal expansion after 5 Hz flicker adaptation, a finding not previously seen in controls. Recent studies have linked genes conferring susceptibility to a cluster of language and sensory deficits to anomalous neural migration, providing a tentative biological basis for dyslexia. However it has proved difficult to establish a clean link between sensory deficits and impaired reading. The results presented here point to an abnormal adaptation response within the early pre cortical stages of the magnocellular pathway, occurring in tandem with a deficit in word level cognitive processing, providing psychophysical evidence for anomalous cortico-thalamic circuits in dyslexia. (Dr. Johnston, Dept Psychology, University College London, Gower Street, London WC1E 6BT, UK)

The results indicate that poor readers in high school may be at high risk for poor saccadic tracking skill. (No corresponding address)

The ability of dyslexic children with or without phonological problems to process simultaneous and sequential visual information was assessed using two tasks requiring the oral report of simultaneously or sequentially displayed letter strings. The two groups were found to exhibit a simultaneous visual processing deficit but preserved serial processing skills. However, the impairment in simultaneous processing was larger in the dyslexic group with no phonological disorder. Although sequential and simultaneous processing skills both related to reading performance, simultaneous processing alone significantly contributed to reading speed and accuracy. These findings suggest that a simultaneous processing disorder might contribute to developmental dyslexia. (Dr. Lassus-Sangosse, Laboratoire de Psychologie et de Neuro-Cognition (UMR 5105 CNRS, Universite Pierre Mendes France, BP 47, 38040 Grenoble Cedex 9, France)

Myopia
from the AAOs Academy Express August 7, 2008. Outdoor Activity Linked to Lower Incidence of Myopia in Children. This population-based study found that higher levels of total time spent outdoors were associated with less myopia and a more hyperopic mean refraction, after adjusting for near work, parental myopia and ethnicity. The authors suggest that light intensity may be an important factor. Pupils would be more constricted outdoors, resulting in a greater depth of field and less image blur. Alternatively, release of dopamine from the retina is known to be stimulated by light, and dopamine can act as an inhibitor of eye growth (Ophthalmology, August 2008)

These data provide further support for local control of emmetropization, as reflected in compensatory lens responses, but point to additional influences on eye growth as reflected in CL-induced ocular changes. (Varuna Padmanabhan, School of Optometry, University of California-Berkeley, 588 Minor Hall, Berkeley CA 94720-2020.

Researchers evaluated the morphologic features, including grade and type, of posterior staphylomas in 108 patients (209 eyes) with high myopia. The prevalence and higher grades of staphylomas were significantly higher in patients age 50 and older, compared to those younger than 50. Overall, type II staphyloma (grade 1) was the most prominent in both young and older eyes; however, in older subjects, the incidence of type II staphyloma decreased significantly while type IX increased significantly. The authors conclude that progression from type II to type IX may increase the mechanical tension on the macular area and optic disc, which may cause the progression to severe myopic retinal degeneration.

Genetic Dissection of Myopia Evidence for
Axial length, a major endophenotype for refractive error, is highly heritable and is likely to be influenced by one or more genes on the long arm of chromosome 5. (No corresponding information)

The Role of the Lens in Refractive Development of the Eye: Animal Models of Ametropia. Sivak JG. Experimental Eye Research 2008; 87:3-8 [Author Abstract]
Research with young mammals and chicks has shown that the visual environment can affect the refractive development of the eye by enhancing or slowing axial eye growth, but the effect on the refractive components of the eye, the lens and cornea, are less clear. A review of the literature indicates that the lens is minimally affected, if at all, and results vary depending on whether the lens is studied in an isolated state or with the accommodative apparatus intact. Research has shown that the development of myopia or hyperopia in young chicks alters lens focal length and magnitude of the accommodative response. However, the result may be indirect or passive due to the effect of the change in size and shape of the globe on the articulation between the ciliary body and lens. Recent research has also investigated the role of the lens in induced refractive error development in a fish, tilapia. Translucent goggles were sutured over one eye for 4 weeks to induce deprivation myopia while the untreated eye served as an untreated contralateral control. In addition to measuring refractive state and intraocular dimensions, a scanning laser system was used to determine the optical quality of excised lenses. All the deprived fish eyes developed significant amounts of myopia and the vitreous and anterior chambers of the treated eye were significantly longer axially than those of the untreated contralateral eyes. No significant change in optical quality was found between lenses of the myopic and non-myopic eyes and the fish recovered completely from the myopia five days after the goggle was removed. The results show that although fish, unlike higher vertebrates, are capable of lifelong growth, the visual environment is an important factor controlling ocular development in this group as well and eye development is not strictly genetically determined. This review indicates that lens growth and optical development is independent from the refractive development of the whole eye. (School of Optometry, University of Waterloo, Waterloo, Ontario N2L 3G1, Canada)

Two procedures to induce experimental myopia, initiated at eye opening, produced significant myopic shifts corresponding to increases in axial lengths after 32 and 46 days of lid suture and after 46 days wearing a -10 D spectacle lens. (Dr. Roger W. Beuerman, Singapore Eye Research Institute, 11 Third Hospital Avenue, #06-00, Singapore 168751, Singapore. Fax: 65-6322-4599)

Approximately 70% of high-risk prethreshold ROP eyes were myopic in early childhood, and the proportion with high myopia increased steadily between ages 6 months and 3 years. Timing of treatment of high-risk prethreshold ROP did not influence refractive error development. There was little difference in prevalence of myopia or high myopia between eyes with zone I and eyes with zone II ROP, nor between eyes with plus disease and eyes with no plus disease. However, prevalence of myopia and high myopia was higher in eyes with retinal residua of ROP than in eyes with normal appearing posterior poles, highlighting the importance of followup eye examinations of infants who had prethreshold ROP. (No corresponding address)

Edited by P.E. Romano, MD, MSO. Abstracts are selected on the basis of interest to our readers. To avoid duplication you will find none are from The American Orthoptic Journal, The British Orthoptic Journal, The Journal of the American Association for Pediatric Ophthalmology and Strabismus, The Journal of Pediatric Ophthalmology and Strabismus, or Strabismus, as most of our readers already subscribe to and/or read them. Publication herein does not constitute endorsement, recommendation or a validation of author’s conclusions.
SURGICAL MANAGEMENT OF STRABISMUS
A Practical and Updated Approach, 5th edition

EUGENE M. HELVESTON, M.D.

Review by David K. Coats, M.D., Houston, Texas

Six pounds of pure muscle; no fat or byproducts here! That’s what the 5th edition of Surgical Management of Strabismus packs. Quintessential strabismologist Eugene Helveston has done it again.

This classic textbook is once again jam-packed from cover-to-cover with all the information that the strabismologist needs to properly plan and execute the management of both simple and complex strabismus disorders.

The text is wonderfully illustrated with step-by-step instructions on how to perform all contemporary procedures that should be in the armamentarium of any serious strabismologist. One of my favorite "extras" in this textbook is a chapter that colorfully explores the history of strabismus surgery from its beginning. What most separates this edition of the textbook from previous editions is the inclusion of an extensive array of case examples complete with histories, clinical photographs, and details of surgical planning. While a few case examples were included in earlier versions, expansion of the case example section in this edition is so extensive that virtually any condition can now be reviewed in detail with a front row seat through the eyes of this world-renowned expert.

Space should be reserved for Surgical Management of Strabismus, 5th edition, in the bookcase of every ophthalmic surgeon. Undoubtedly this reserved space will be vacant most of the time, as this book is most likely to remain open and in constant use on the surgeon’s desktop.

THE BOOK
The HISTORY OF STRABISMOLOGY is the first monograph devoted entirely to the development of strabismology in different regions of the world. Each of the co-authors has been assigned a special chapter in which his or her knowledge of the material is particularly profound. The origins of strabology go back to the beginning of medicine, thousands of years ago. The story how this specialty evolved from quackery and superstition in ancient times to its present state of sophistication is a fascinating one. It should be of more than passing interest, not only to those specialized in this field but also to others with an interest in the history of ophthalmology.

The book consists of approximately 400 pages and is abundantly illustrated with fine reproductions of old documents, engravings, drawings and historic instruments, many of which are from ancient and rare manuscripts. Printed on deluxe art paper THE HISTORY OF STRABISMOLOGY is bound by hand and gold embossed on book plate and spine.

THE EDITOR Gunter K. Von Noorden is a world-renowned author and strabologist. His expertise in the entire field of strabismus is documented in his textbook (now in its 6th edition) and uniquely qualify him to organize and edit a book on the history of strabology.

THE AUTHORS The authors are prominent strabologists from different parts of the world, internationally known for their contributions. Indeed many have actually played an active part in shaping the history of strabismology during the second half of the 20th century. They are joined by a comprehensive ophthalmologist who is also an ophthalmic historian of international reputation and by one of the leaders of the orthoptic profession. The following contributed to this book: Henderson C. Almeida, MC, Shinobu Awaya, MD, Alberto Brown-Limon, MD, William E. Gillies, MD, Eugene M. Helveston, MD, Joseph Lang, MD, Emma Limon de Brown, MD, Gunter K von Noorden, MD., Hans Rmeky, MD, Geraldo Ribeiro de Barros, MD, and Gill Roper-Hall, DBOT, CO, COMT.
MULTIMEDIA REVIEWS

LEE M. JAMPOL AND ANGELO P. TANNA, EDITORS


To quote Dr. Kushner, “Many roads lead to orthophoria.” The treatment of strabismus, that “art form” with scientific underpinnings, can be the bane or the joy, or more likely both, of the ophthalmologist’s existence. For those of us in pediatric ophthalmology, it is our “bread and butter” and the source of endless discussions and debates. There is enough science to provide a logical approach, and enough art to make things really interesting.

This book is a compilation of 68 cases published over 17 years in the journal Binocular Vision and Strabismus in its “Grand Rounds” section, edited by Dr. Kushner. The cases are presented in a standardized format, including summary of the therapeutic problem, history, eye exam, and final diagnosis. Dr. Kushner states that he was not attempting to solve a clinical problem for a specific patient but rather presenting an intellectual exercise with input from respected colleagues. Indeed, he does not present his own opinion, nor the actual results following treatment on most of the cases. Several experts in the field present opinions on the diagnosis and treatment. Each case is followed by the editor’s perspective which highlights the issues raised. The cases cover clinical topics from nonparalytic vertical strabismus to cataract. There were 249 different individuals who served as discussants for one or more cases. This provides a broad perspective covering many schools of thought.

The cases are numbered and have descriptive titles such as “A Case of ‘V-pattern’ Esotropia with Excyclotropia after Bilateral Superior Oblique Tucks.” These allow for easy selection of cases for clinical purposes or teaching. The cases are interesting and informative analyses of complicated problems, primarily involving strabismus. However, although it is useful to have these case reports, previously published in a journal, collected together in one volume, it would have been more useful to have included outcomes and follow-up. Nonetheless, the compilation provides a thought-provoking read, an aid to clinical problem-solving, and a stimulating jumping-off point for teaching sessions.

Marilyn B. Mets, MD
Chicago, Illinois, USA
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(from Investor’s Business Daily Nov. 26, 1997 by M. 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 readers 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’. ... 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).

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D. BRIAN STIDHAM MEMORIAL LECTURESHP

LECTURE to be published annually in Binocular Vision and Strabismus Quarterly

Donations Solicited 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.

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

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Kenneth W. Wright, USC Keck School of Medicine, Los Angeles, CA, USA

Color Atlas of Strabismus Surgery

Strategies and Techniques

Color Atlas of Strabismus Surgery: Strategies and Techniques provides concise, comprehensive descriptions of surgical procedures by one of the world's leading experts. The accompanying DVD brings the book to life with real time, narrated video of the procedures. Dr. Wright's narration not only explains the procedures, but provides pearls and pitfalls to allow for the best possible patient outcomes. Pediatric ophthalmologists, ophthalmology residents and fellows, as well as general ophthalmologists, will find this atlas to be essential to their work.

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