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ROMANO: Epigenetic Myopia; Epipha-Me; Globe Fix: IOA iii

MIMS III: HISTORY of MEDICINE: Ocular Disorders of the Mona Lisa and Other Famous Paintings

*** ORIGINAL EVIDENCE-BASED SCIENTIFIC ARTICLES ***

AKBARI, AMOLI, ALHASHEMI, AMERI, JAFARI, ESHRAGHI and BOZORGHI: Histological Changes by Bupivacaine Injection on Rabbit Extraocular Muscles

HESHAM, SIMON JW and ZOBAL-RATNER: Left-Sided Predominance in Pseudoesotropia

ROMANO: Stage III [End-operative] Intraoperative Adjustment of Eye Muscle Surgery [for binocular re-alignment] for Neuroparalytic and Mechanical Incomitant Strabismus

*** CASE REPORT With Management and Outcome ***

AKBARI, JAFARI, AMERI, ANVARI, ESHRAGHI & MASOOMIAN: Successful Re-Resection for A "Snapped" [Severed, Inadvertently] and Retrieved Inferior Rectus EOMuscle

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HYDE PARK EDITORIAL: Consciousness: A Function of Stereoscopic Vision Via the Egocenter. = the Binocular: Its where we live

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“... the belief that one’s view of reality is the only reality is the most dangerous of all delusions ...”-Watzlawick, 1976

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Losses. Obituaries.

Yes we are living longer thanks to all those expensive advances in our field of medicine, but nobody is immortal yet. Considering the direction the rest of the world outside of medicine is heading... we don’t really want to be, that is for sure....

Just a year ago we (BV&SQ) lost one of our biggest supporters, renowned Australian orthoptist, Zoran Georgievski. He was a proponent of the International Orthoptists having their own scientific journal, including maybe adopting this publication. But when he passed on, so did that idea.

Then suddenly and much too soon another former BV Ed Board member and early supporter, all too young, John P. Lee. Now getting caught up on more bad news.

One of our original supporters, Bruno Bagolini, of Striated Glasses fame, who also helped our mentor Gunter von Noorden so much..... Passed away too.

Here are his contributions to BV&SQ:

Bagolini Lenses: ERRONEOUS TERMINOLOGY!
See Bagolini Striated GLASSES.

Then fellow AAPOS member and leader, Michael Redmond, a victim of Steve Jobs’ disease, passing similarly.

And now also another of BV&SQs early and strong European supporters, Joseph Lang... of Micostrabismus and Autostereoscopic testing fame and popularization of the term “strabology”...

Joseph was a great supporter from our very conceptual start and he especially contributed to the publication’s acceptance by the European strabology community.

These are his contributions to the pages of BV&SQ:

LANG J: An efficient treatment and new criteria for cure of strabismic amblyopia: Reading and Bangerter Foils. 1999; 14:9
LANG J: Congenital or infantile: That is the question. 1988; 3:116 (Hyde Park Essay)
LANG J: Stereopsis assessment in normal 1.5 to 3 year old children: A comparison of three random dot tests: Operant preferential looking; Random Dot E; and the Lang Stereotest I. 1996; 11:241 (correspondence)
LANG J: The historical Microtropia semantic debate continues after a third of a century. 2000; 15:313

Lang Stereotest:
Assessment of adult stereopsis using the Lang 1 Stereotest: A pilot study. 2001; 16:91
Stereopsis and visual acuity: Their combined importance in eye screening preschool children. 1990; 5:65
Stereopsis assessment in normal 1.5 to 3 year old children: A comparison of three random dot tests: Operant preferential looking; Random dot E; and the Lang Stereotest I. 1996; 11:125

We most enjoyed and appreciated the Lang obituary story in the J AAPOS at the end of last year which we quote here....

From J AAPOS 2011; 15(6) December 2011 by Albert Franceschetti, M.D.

“... His influence was mainly felt through his work Strabismus, ...of such exceptional interest that it was widely translated. His [most
successful] contribution to the field was the [auto-]stereoscopic test named after him, which was enthusiastically adopted by pediatricians and ophthalmologists. It enables testing of stereoscopic vision, even in toddlers, at 1200, 600 and 550 seconds without green-red glasses” [or any spectacles, at all, a huge advantage in children].

“Dr. Lang’s research also led him to discover microstrabismus, a previously unrecognized and untreated condition. He announced his discovery in 1970 at the Congress of the International Strabismological Association (ISA) in Acapulco, Mexico. His North American colleagues, however, insisted that they had discovered it first. I still remember swimming in the hotel pool with Joseph, screaming at the top of his lungs in Swiss German, ‘They have stolen my microstrabismus!’ . Fortunately, this accomplishment was eventually acknowledged as rightfully his.

…”

We do and shall miss all of these, our fellow strabologists of a lifetime. - per
EDITORIAL: EPIGENETIC MYOPIA: Rx Control It!  
EPIPHA-ME  Globe Re-Fixes with IOA III.

We start this year and this first editorial with a departure back into one of my favorite pediatric ophthalmology subjects: myopia, especially that of the so common eye disease, “school” myopia.

When we studied and treated myopia way back when, we didn’t know anything about epigenetics. We proselytized that it could and should be prevented and treated right then with atropine eye drops (and appropriate progressive add, dark spectacle glasses, brimmed hats.

The response from too many was “myopia is a genetic disease. You cannot and better not try to treat it with eye drops! You have to fix the GENES! It needs genetic treatment of some sort which we just haven’t figured out yet.” A former colleague pushed me out of his web site for the strength of my persistent preaching on myopia control.

At the Toronto Meeting of the AAPOS in 2000, (one week after I had a stroke), we gathered a dozen or so members together who thought the idea was a good one. We proposed to form POMMM, “Pediatric Ophthalmologists for the Medical Management of Myopia”... and there was a positive consensus and lots of interest in studying atropine further, but we couldn’t raise the funds and our multi-institutional PO research group was in its infancy then...

But now there is epigenetics. My attention to this was captured and enhanced by an article in a recent National Geographic (1) “…nature and nurture are not the only elemental forces at work. According ... to epigenetics there is a third factor also in play,

“...one that in some cases serves as a bridge between the ENVIRONMENT (ed.emph.) and our genes, and in the other operates on its own to shape who we are.” (And that would include ALL aspects of the visual environment and its manipulation.)

Twin studies are the source of this, especially since “1980, following the discovery of numerous identical twins who’d been separated at birth.”

NG gives a couple of glaring examples of epigenetics: identical twins, female, one of whom has early onset Alzheimer’s, and is totally disabled while the other is entirely normal. And:

Two identical twin boys who are both autistic but at opposite ends of the spectrum. The one who is impaired the most also had congenital heart disease and spent his first 6 months in hospitals and surgery. The other, a voracious reader, needs glasses (but NG and they missed? that evidence of epigenetics!)

Epigenetics is NOT really new although the term is. We used to just call it the “variable expressivity” of genes. Twin studies have now provided hard evidence for it and loosened the iron hold of geneticists on our lives!

A more specific example of non-twin epigenetics is given in Wikipedia: in rats licking and grooming their pups a lot gives rise to an escalade of beneficial gene options.

After reading in this article how much DNA does determine, I feel as if I have lost any “free will” that I may have thought I had.
Too, so many things are pre-determined by your DNA, even if many are also part determined by nurture and the environment.

AND it does leave plenty of room to say that, since we DO know how immense the effects of environment are on the development of myopia in all sorts of animals, not just man, we think there is a good case that it is primarily an epigenetic disease, not at all strictly a genetic one, as so many have insisted... (see also references below)

AND myopia is controllable or even reversible as we found it (2), with as little as a single dose once a week (3).

AND NOW, even with minute [almost homeopathic?] doses of atropine *, (once daily as low as only 0.01% atropine) (4).

Authors Conclusions: “Atropine 0.01%, administered once nightly to both eyes for 2 years, has minimal side effects compared with atropine at 0.1% and 0.5%, and retains comparable efficacy in controlling myopia progression.” (Ed bolded)

I still would recommend using all my anti-retinal toxicity steps (5) but this dose reduces those risks another hundred fold!

So you can go right ahead and treat school myopia as aggressively as we did way back then (even including pushing outdoor activities(6) and Progressive Adds which now have been cleared of the terribly remote possibility of rebound (7)). Myopia remains a serious disease: dangerous sequelae: it doubles the risk of open angle glaucoma (8).

Editorial Followup from last issue:
“LIKE”> on the ‘net:
From BV 26(1): “IT IS “CLICKS” (I.E., “LOOKS” AND “LIKES”) THAT

(Courtesy National Geographic)
COUNT- NOT SALES OR EVEN WHO BUT to HIT “FRIENDS” BACK AGAIN to fluff their numbers’. And EGOs.

UPDATE THURSDAY FEBRUARY 02, 2012: The FACEBOOK IPO IS OUT TODAY: WSJ: “The power of ‘LIKE’ as an emotional sensor is driving the company’s exorbitant evaluation.” [even if devised in 2010, it is today:] “The Button That Made Facebook Billions” [$75-100 Billion!...by Andy Kessler p. A13.

And the latest election 2012 tip is that the ones we choose to actually and finally vote for are those that “we like the best” and all other factors are trivial, useless... B.O. IS like... all charisma & silver tongue!

So that even at the highest level of our meritocracy?, popularity, rather, and finally, is all that counts... January 26, 2012:

From “Facebook Obsession”, a CNBC feature, it is revealed that this whole “Like” FAD was contrived by Facebook just to provide more detailed info on participants for their advertisers, to concentrate their ad dollars and efforts on their most likely customers. Once again it’s “sell, sell, sell!!!” But talk about viral....

But Facebook has been too successful.

Seems like everything in the world now comes down to that single word or button, “like”.

A teen girl locally, invented a new word: talking about the epiphany SHE had, she called it an epipha-ME instead... Right on!

As much as we personally abstain this new fad, it also occurs to us that this might lead to a real change in the world we live in, something of the magnitude of Christianity:

Suppose as a result everybody decided being liked was really the most important thing about life?????? And really devoted themselves to accomplishing that.. No More Hatred or Killing? DREAMER!!!!

Here’s reality:

**In this Issue**

Bupivacaine Injection Myotoxocity on Extraocular Muscles. A Strabismus Alternative Treatment: Extended Histological Changes Induced in a Rabbit Model. Akbari MR,

Chemodenervation again but with a big twist, now hoping to take advantage of the implied increase in extraocular eye muscle contraction and binocular alignment potential which occurs with this drug in rapid recovery phase ... These workers confirm prior studies but over a more extended study time.

We look forward to more reports on this promising new avenue of therapy for strabismus and other extraocular motility problems.


We are again indebted to these authors for another major review of pathology of binocular vision and strabismus. This one is limited to the diagnosis and initial and/or early non-surgical management of these difficult clinical situations.

The surgical management of these is planned for the next and third review in this series which we hope to publish later this year.

Perhaps they will be able to update the stage III (end-operative) intraoperative surgery adjustments for such cases that we have printed in later pages of this issue (pages 46-50). This sector of our work is yet another place that maybe could use it.

**Ocular Disorders of the Mona Lisa (Strabismus) and Other Famous Paintings in the Louvre, Paris. Mims III JL. *Binocul Vis Strabolog Q 2012; 27:35-38*  

The Mona Lisa ... I always thought that her charm was all in that enigmatic sort of smile of hers... asymmetrical, curved up on the left, pretty flat on the right. Take the edge of a good mirror and place it on the sagittal center of her face if you want to see how different she looks when you make either side symmetrical with a mirror... did she maybe have a little right seventh nerve paresis too, in addition to her Duane’s Syndrome?? I am not aware of the coincidental occurrence of that situation... PubMed doesn’t have any immediate reports of that coincidence...

And you won’t find the ace here!.. It got clipped in taking the picture of the artwork. But the warnings of this allegory picture are very out there: gambling, wine and women; watch out!


Would someone please supply the Academy with a picture of pseudoET which does NOT arouse suspicions that it might not be?. The corneal light reflections totally confuse the issue (and that diagnosis)! *[but not for Mom...]*

We did especially appreciate the authors’ hypothesis to explain what they found on a regular basis, so you may also expect to observe this in your patients.


Yet another article on “restrictive” incomitant strabismus, following the two (plus) articles and ten cases we had in the last issue (and at least one more to come in the next issue). This is just one more case, but thoroughly discussed.

*First some more words on Words as in the last issue. There, we carried on a bit about a “slipped muscle”. We again attacked it as we had in a an editorial 8 years ago in issue 19(3)137-8.*
We went further: It should be “Strabismus Surgery Complication: Suturing/suture material failure suspected.” or “Failure of surgery” (This term, “slipped” muscle, is... a cover-up: to shift... fault and blame...[for] error... to an inanimate and defenseless third party: the extraocular muscle of the patient... shifting the blame... TO THE PATIENT! This SHIFTING BLAME TO THE VICTIM is “S.O.P.” today...

“ In our highly litigious society and there are many examples, especially in government where political guilt must be totally avoided, as well as in medicine where lawsuits must be avoided. Here, a surgeon has SLIPPED, NOT the muscle ! (Think that instead...)”

So we have yet another example with this case: Here it is a “snapped” muscle. Everything we said about “slipped” applies totally equally here. So we added two more terms into the title.

The authors have done a thorough job with their report; they were just following conventional poor strabology “English”.

Second: All these reports mentioned at the start of this comment are for seriously incontinent complicated strabismus problems for which there are few if any guidelines for surgical correction and realignment of the eyes.

None mention the technique we used so successfully in such cases. It was in a report I gave as a poster at an ISA meeting and was only in the transactions of that meeting. So we have now published it here:


These are all cases of compound monocular binocular ocular misalignment. How’s that for a term describing an alignment problem primarily in the movement and/or absolute position of one eye, but this contributes to a binocular misalignment and both problems need remediation, but together. That is why it is so and most difficult to determine exactly what to do and especially how much to do, to get the best binocular alignment for the patient....

Our results reported are better than those in the aforementioned group of papers on “restrictive strabismus”. And all the principles of our IOA system are confirmed in the most recently published papers using our IOA techniques. (See article proper [pages 46-50] for those references.)


Hope to see all in San Antone. Have safe trip.
Bupivacaine Injection Myotoxicity on Extraocular Muscles. A Strabismus Alternative Treatment: Extended Histological Changes Induced in a Rabbit Model

MOHAMMAD REZA AKBARI, M.D., FAHIMEH ASADI AMOLI, M.D., LEILA HOSSEINI ALHASHEMI, M.D., AHMAD AMERI, M.D., ALIREZA KESHTCAR JAFARI, M.D., BAHRAM ESHRAGHI, M.D. and SHIMA BOZORGHI, M.D.

from Farabi Eye Research Center, Tehran University of Medical Sciences, Tehran, Iran

ABSTRACT: Purpose: To evaluate longer term histological changes of extraocular muscles induced by bupivacaine toxicity.

Methods: The superior rectus and inferior rectus of white rabbits were injected with 0.4 mg of agent in 0.3 ml of bupivacaine. Then, histological changes of these rectus muscles were examined at 1, 2, 4 and 8 weeks after injection.

Results: Bupivacaine injection induced myotoxic changes in both orbital and global layers. Regenerating myofibers were found at 1 week after injection. These changes were reduced by 4 weeks post injection. At 4 weeks increasing muscle fiber size in both orbital and global layer were seen. There was no scar formation after 8 weeks post injection.

Conclusions: Extraocular muscle bupivacaine injection can cause acute myonecrosis followed with regeneration. After 8 weeks the muscles recovered with arranged myofibers almost at normal level. However we found increased myofiber diameter at 4 weeks after injection which was remained stable until 8 weeks post injection. Further investigation about functional change in these muscles is needed to facilitate application of this methodology.

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INTRODUCTION

The extraocular muscle (EOM) of mammals has unique characteristics compared to other skeletal muscle, which are composed of two layers: a central global layer containing larger diameter fibers, which continuous to its tendinous insertion in the sclera, and a peripheral orbital layer, consisting of smaller muscle fibers that insert in the corresponding pulley of the rectus muscle (1,2).
Strabismus, an imbalance in the action of the extraocular muscles, can be treated surgically or pharmacologically. Myotoxic agents can be useful in the treatment of strabismus.

For the first time, Botulinum toxin A was clinically used in 1980 by Scott; however, short duration of action, effects on adjacent EOMs and blepharoptosis limit its usefulness (3-5).

Eye muscles are also sometimes injected with bupivacaine during retrobulbar or periocular anesthesia for cataract and other intraocular procedures (6,7). Postoperative strabismus usually manifests by muscle paresis followed by overaction (8). Recently Scott and coworkers used bupivacaine injection in extraocular muscle to treat esotropia. They believe the muscle's response after retrobulbar anesthetic injection for eye surgery lead to misalignment or strabismus. So this muscle's response can use successfully to improve eye alignment and correct strabismus (9,10).

Carlson and colleagues demonstrated that direct injection of bupivacaine induced rapid degeneration and regeneration of EOMs in monkeys (11).

In this study we examined histological changes in rabbit EOMs induced by bupivacaine injections for longer periods than previously reported.

MATERIALS and METHODS

This study was done between December 2010 and February 2011 in Farabi Eye Hospital of Tehran University of Medical Sciences designed in compliance with the guiding principles in the care and use of animals.

Twenty aged New Zealand white male rabbits (2-3 kg) were anesthetized with an intramuscular injection of ketamine and xylazine, 10mg/kg and 2mg/kg, respectively.

After superior and inferior conjunctival peritomy superior and inferior rectus were exposed and 0.4 mg in 0.3 ml of bupivacaine was injected into each muscle at 10 mm posterior to insertion, using a 30-gauge needle. Large areas of swollen muscle tissue were noted at injection sites immediately after injection.

Three rabbits were euthanized with an overdose of xylazine at 1 week and 12 rabbits (4 rabbits in each group) were euthanized with an overdose of xylazine at 2, 4 and 8 weeks after injection then superior and inferior rectus muscles were removed from origin to their insertion (2 eyes in each rabbits, 2 muscles per eye).

In control group I, after superior and inferior conjunctival peritomy and exposure of rectus muscles 0.3 ml saline were injected in right eyes of 3 rabbits without injection in their left eyes. These rabbits were sacrificed at 1 week after injections.

In control group II, 0.3 ml saline were injected in superior and inferior rectus muscles of both eyes of 2 rabbits, then these muscles were removed after 8 weeks post injection for histological examination.

The excised extraocular muscles (with a suture for orientation for cross sectioning of each muscle) were fixed in formalin and were processed for paraffin embedding. The sections were stained with hematoxylin -eosin and masson trichrome. In each muscle multiple sections from origin to insertion were processed, the section showing the most prominent changes, which was near the injection site was selected for histological evaluation.

RESULTS

80 muscle specimens of 40 rabbit's eyes were processed for histological evaluation. In study group myotoxic changes including inflammatory cell infiltration, myofiber destruction and fibrous tissue formation between cells were present at 1 week after injection in both global and orbital layers.
Regenerating myofibers with coarse staining, pale nucleus, central migration of nuclei and increasing diameter in orbital layer and decreasing diameter in global layer stained with hematoxylin-eosin were prominent 1 week after injection. See Figure 1. Regenerating myofibers were rarely found at 4 or 8 weeks post injection. Inflammatory changes decreased over 2 weeks.

**Figure 1 (Akbari et al): Rabbit extraocular muscle. Changes at 1 week after injection (hematoxylin-eosin). A. In both control groups with and before injection of normal saline, the normal-appearing muscle fibers arrangement. B. Under low magnification (×100) areas of regenerating muscle fibers with coarse staining, pale nucleus, central migration of nuclei, multinucleated cells and inflammatory cell infiltration with bupivacaine injection. C. Under high magnification (×400) multinucleated myofibers, central migration of nucleus are shown. D. Area of injured muscle tissue degeneration and myofiber destruction after bupivacaine injection.**
At 2 weeks after injection increasing inter-myofiber space was detected. At 4 weeks after injection myofiber arranged to almost normal level in both layers. After 4 and 8 weeks post injection increased myofibers diameter in both global and orbital layers were present compared to control groups. At 8 weeks post injection congestion of sections were detected. There was no scar formation 8 weeks after injection. These findings are shown in Figure 2. In the transverse

**Figure 2 (Akbari et al):** Hematoxylin-eosin staining of rabbit extraocular muscle at 2, 4 and 8 weeks after injection of bupivacaine under low magnification(×100). **A.** Notice increasing inflammatory cells, however few regenerating myofibers still present after 2 weeks post in inter-myofiber space, post injection of bupivacaine. **B. and C.** At 4 and eight weeks after injection myofibers recovered to almost normal arrangement. **D.** Congestion of vessels is prominent at 8 weeks post injection.
section of the most severe changes, we randomly select 50 myofibers for the diameter analysis and 100 myofibers for the proportion of centrally nucleated myofibers. Myofibers diameter were measured by Zeiss microscope Axiosstar plus and ocular micrometer.

In the control groups, the diameters of the myofibers in the orbital and global layers were 8±2 µm and 18±4 µm respectively.

Bupivacaine injection induced an increase in myofiber diameter in the orbital layer at 1, 2, 4 and 8 weeks after injection (10±4 µm, 11±4 µm, 12±4 µm and 12±4 µm) versus control. (P=0.08, P=0.04, P=0.045, and P=0.045 by paired t-test)

In the global layer myofiber diameter was reduced at 1 week after injection 15±6 µm (P=0.036 by paired t-test), normalized 2 weeks after injection 18±5 µm (P=0.08 by paired t-test) and increased at 4, 8 weeks after injection (20±4 µm, 20±8 µm)(P=0.04 and P=0.048 by paired t-test) shown graphically in Figure 3, below.

One week after injection 20 % of the orbital and 15 % of the global myofibers, contained centralized nucleus. Centralized nucleus myofibers decreased over time. However, less than 1% of the myofibers were centrally nucleated at 4 and 8 weeks after injection. The features of regeneration and variable myofibril size were more prominent in the orbital layer 1 week after injection.

In the control groups, we had no histological changes including degeneration or regeneration of myofibril either in injection of saline or without injection groups. These histological changes are summarized in the Table, top next page.

Figure 3 (Akbari et al): Graphic representation of Bupivacaine injection induced increased myofiber diameter in orbital layer and decreased myofiber diameter in global layer of extraocular muscle 1 week post injection. Finally, myofibers diameters were increased in both orbital and global layers at 8 weeks after injection.
DISCUSSION

In this study, we present histological and myofiber size changes after intramuscular injection of bupivacaine in both orbital and global layers of rabbit extraocular muscle.

Bupivacaine (an aminoacyl anesthetic) injection can induce cell death and myonecrosis by intracellular increase of calcium level (12). In previous animal studies Z-band lysis and sarcomere dissolution at the Z-band have been shown to result in the loss of contractile function within minutes of injection (13). The damaged cells are removed by macrophages, leaving the cell membranes, nerves and blood vessels intact (14). Then autocrine growth factor molecules released from damaged area activate satellite cells that proliferate new muscle fiber to replace the damaged muscle (15). Satellite cells play an important role in muscle regeneration, with maximum activation of satellite cells occurring 2 to 3 days after injection.

The peak of regeneration occurs 3 to 4 days after injection and can persist for up to 2 weeks. Studies have shown that striated muscle tissue regeneration is usually complete within 4 to 6 weeks (16). In another study, the rat limb...
muscle regeneration via muscle fiber remodeling processes was reported by 60 days (17).

In this study, degeneration myofibril, myonecrosis and inflammatory cells infiltration followed by regenerative changes including central migration of nuclei, multinucleated cells and increasing diameter in orbital layer and decreasing diameter in global layer were prominent 1 week after injection as in other studies. However these changes were more prominent in orbital layer; in contrast to Park study which these changes were more prominent in global layer. Although there is still the possibility that differential effect in orbital and global layers is due to diffusion differences after an injection, both the different myofiber composition between the two layers and the unique characteristics of the myotoxin may play a role in the predilection of the damage (18).

At 2 weeks after injection, inflammatory changes decreased and myofibril diameter normalized in global layer and increased in orbital layers. Increasing myofibrils diameter continue . At 4 weeks after injection myofibril diameter increased in both layers. In present study we observed increasing both layers myofibril diameter. Additionally, these findings were constant until 8 weeks post injection in this study.

In the Park et al study, the same results were presented at 1 week after injection but myofibril diameter normalized 4 weeks after injection in the global layer and increased in orbital layer. However their study was done ONLY up to 4 weeks post injection (18).

The areas of muscle changes were more prominent near injection site in this study, consistent with other studies (8,18).

McLloon and colleagues demonstrated that skeletal muscles formed new myofibers through the activation of progenitor cells, satellite cells and myofibroblast. They found large influx of acute inflammatory cells at 1 day after injection followed by rapid degenerative phase, lasting approximately 2 days. The peak of regeneration occurs 3 to 4 days and persist for up to 2 weeks and usually complete within 4 to 6 weeks (16,19-22). Consistent with other studies, our study revealed acute myotoxic changes including inflammatory cells infiltration and myofibers destruction and regenerating muscle fibers with features of immature cells. These findings were found to peak at 1 week post injection and rarely occurred later (20,23).

Previous studies reported complete extraocular muscle recovery after bupivacaine myotoxicity, however Zhang et al reported 50% of focal areas of fibrosis, representing variability of response (8). Bleik and colleagues showed a clinical specimen with atrophic inferior oblique muscle fibers with surrounding fibrotic tissue after local anesthetic injection (24). In this study, in contrast to areas of myodestruction at 1 week post injection, there was no area of fibrosis 8 weeks after injection.

Recently, Scott reported increasing the average muscle size and hypertrophy (10). Furthermore, the modeling program-“orbit 1.87M” indicate muscle hypertrophy, not shortening or stiffening from scarring, and this can explain the muscle overaction pattern characteristic of these cases (25). He (Scott) treated esotropic patients with a high concentration bupivacaine injection into the lateral rectus muscle (9-10).

In conclusion, we found complete recovery of extraocular muscle and increased both layers myofibril diameter of rabbit extraocular muscle after intramuscular injection of bupivacaine which was constant up to 8 weeks. Despite the histology and morphometrical value, the physical characteristic, such as contraction of these recovered muscles warrants further investigation. If this hypertrophic muscle continues on to build a muscle having greater contractile strength than before ,with consequent effects on binocular eye alignment, we can use
this muscle response to improve or remedy binocular eye misalignments.

REFERENCES


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ABSTRACT: The purpose of this presentation is first to describe the symptoms and problems encountered in cranial nerve palsies (CNP). The purpose is also to describe the different means of treatment during the observational preoperative period and their positive or negative impact on each of the symptoms and problems. Finally, we will present our way of handling these patients in their preoperative period: practical, inexpensive, and unsophisticated means that keep the patient comfortable and prevent the secondary untoward effects that can take place.

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OUTLINE

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C. Our Way of Managing CNP in the Acute Phase and During the Observational Period
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A. Symptoms and Problems in Cranial Nerve Palsies (CNP)

I. Diplopia
   The diplopia is due to the images of the objects of regard in the “area of conscious regard” falling on non-corresponding retinal areas with two different visual directions [1].

II. Abnormal Head Posture (AHP)
   a. Fusional Compensation
      A patient with incomitant strabismus will adopt a head posture to obtain single binocular vision (SBV). He will, by reflex mechanism, find a zone where there is SBV. In eye muscle palsy, the AHP always aims at keeping the muscle at fault “at rest”, and stimulate normal muscles.
      • In cranial nerve VI (CNVI) Palsy, the head turn is to the side of the palsied lateral rectus (LR) muscle, keeping the eye away from the LR field of gaze.
      • In medial rectus (MR) muscle palsy, the head turn is to the contralateral side in order to keep the eye away from the MR field of gaze.
      • In vertical rectus muscle palsy, the AHP could vary between head turn, and/or abnormal chin position, and/or head tilt:
         i. Head Turn: Occasionally seen, placing the eye in the field of lesser deviation [2] –usually
abduction in oblique palsy and adduction in vertical rectus muscle palsy, since the maximum vertical action of the oblique is in adduction and the maximum vertical action of the vertical recti is in abduction.

ii. Chin Depression: In V-pattern esotropia, chin depression is commonly seen, placing the eyes in upgaze to avoid the esodeviation in downgaze and thus allow fusion in upgaze. Sometimes it is used to avoid large vertical deviations in downgaze.

iii. Chin Elevation: Likewise, chin elevation is expected in V-pattern exotropia to avoid exodeviation in upgaze, and to allow fusion in downgaze. Sometimes, it is used to avoid large vertical deviation in upgaze.

iv. Habitual Head Tilt: It is usually an effort to avoid activity of a paretic muscle so that the tonic impulses sent by the otolith apparatus do not have an effect on that muscle. It is very common in cyclovertical muscle palsies, especially in oblique palsy, since the obliques are the primary torters of the globe (60% torsional action compared to 40% vertical action). Habitual head tilt is not encountered in MR/LR muscle palsies, unless a cyclovertical component takes place due to secondary oblique anomalies, dissociated vertical deviations (DVD), or latent fixation nystagmus. It can also happen secondary to a disturbance of the tonic vertical vergences, or tonic cyclovergences due to disruption of fusion from the palsy.

Head tilt has been considered variously as a compensatory posture to decrease the torsional effect and as a compensatory posture to decrease the vertical effect. We have measured the difference between the vertical deviations with the head tilted to the right and to the left, and the difference between torsional deviations with the same head tilt, in 8 cases of superior oblique (SO) palsy[2]: the average difference between the vertical deviations was 15 prism diopters (PD), while the difference in torsional deviations was only 2 degrees. This marked difference of the vertical deviations with head tilt to one side as opposed to the other side, and the very modest difference in torsional deviations lead us to believe that an habitual head tilt in cyclovertical muscle palsy, is usually a compensation to reduce the vertical deviation and thus gain fusion. In addition, if in a patient with a combination of vertical and torsional deviations, one neutralizes the vertical deviation with prisms, the AHP disappears[3]: This also shows that head tilt in cyclovertical muscle palsies correlates with the vertical deviation rather than with the torsional deviation.

b. Balancing the Passive Mechanical Forces.

Another not uncommon cause of AHP is restrictions, especially when these restrictions are operative in the primary position. With imbalance of mechanical forces in the primary gaze, fixation must occur under stress and duress: the excessive innervation for the task will thus cause an overdrive of the fellow eye, which may –or may not- be compensated for by the fusion mechanism. In such a case, in order to fixate with less duress against the restrictions and better mechanically balance the fixating eye, a compensatory head turn or chin position is adopted. The result is always to keep the restricted muscle in its own field of gaze (MR in adduction, LR in abduction, SR/IO in elevation, IR/SO in depression).

c. Balancing the Active Forces

Imbalance of active forces between agonist/antagonist muscles is another common cause of AHP. It is mainly encountered in muscle palsies and co-contraction syndromes, such as Duane Syndrome (DS).

In muscle palsy, there is –in the primary position- an imbalance between the palsied muscle and its normal antagonist. In co-contraction syndromes, the imbalance is between the anomalously innervated muscle –that is a muscle receiving innervation from a cranial nerve that does not normally innervate that specific muscle- and its normally innervated antagonist [4]. The patient will compensate by turning his/her head in order to find a zone –not necessarily extreme gaze away from the primary position- where there is an innervational balance between the two antagonist muscles. That is why, as Jampolsky pointed out, a large compensatory head turn for fusion may denote a mild Duane Syndrome or a mild palsy and contrariwise, a small head turn paradoxically usually denotes a more severe Duane Syndrome pattern [4] or more severe muscle palsy.

In co-contraction syndrome, the compensatory head turn is always to the side of the affected eye in
Duane esotropia, and always to the opposite side in Duane esotropia. In muscle palsies, the aim of the AHP is to keep the affected muscle “at rest” and stimulate normal muscles. In LR and MR muscle palsy, the head turn is always to the side of the affected muscle in order to keep the eye in adduction in LR muscle palsy, and in abduction in MR muscle palsy. In cyclovertical muscle palsy, the head turn is less common and, when it takes place, its purpose is not to balance the active innervational forces, but to place the eye in the field of lesser vertical deviation.

III. Binocular Misalignments: (Strabismus) Esotropia/Exotropia, Hypertropia and/or Hypotropia, Incyclotropia and/or Excyclotropia

In unilateral cases, if the palsy is partial and mild, the patient usually maintains his/her binocular fusion in the primary position and in the gaze opposite that of the paretic muscle. However, binocular fusion is completely disrupted in the field of action of the paretic muscle. In complete paralysis, fusion is usually disrupted in the field of action of the paralytic muscle and in the primary position. It may even be lost in the gaze opposite that of the paralytic muscle.

In bilateral cases, fusion is disrupted in all fields of gaze. In congenital and longstanding cases, fusional amplitudes of convergence/divergence, sursumvergence/deorsumvergence, or incyclovergence/excyclovergence can expand to large amounts, resulting in a deviation kept latent by fusion with an AHP (head tilt and/or head turn and/or abnormal chin position).

There commonly is an association of horizontal, vertical and torsional deviations in cyclovertical muscle palsies and even in CNVI palsy.

Vertical and torsional deviations in CNVI palsy are common, suggesting that the CNVI and the LR muscles play a role in the vertical alignment of the eyes [5]. These deviations can also be due to disturbance of the tonic vertical—as well as the tonic torsional- vergences, resulting in “sensory vertical or torsional deviations” [6].

Vertical and torsional deviations in CNVI palsy can also be due to the recruitment of the secondary abduction action of the SO muscle to help the deficient abduction. Lastly, secondary vertical and torsional deviations can be due to the hypertonicity of the SR and the SO muscles.

The diagnosis in CNP can be obscured by the spread of comitance (SoC), common in longstanding cases. SoC is due to innervational factors spreading to the other extraocular muscles (EOM): direct antagonist and yoke, the inhibitional palsy of the yoke of the antagonist [7], as well as to the synergistic hyper and hypo muscles [8,9]. This spread of innervation results in mechanical factors due to muscle-length-adaptation characteristics of EOMs with shortening of some muscles due to actual loss of sarcomeres and lengthening of others due to actual gain of sarcomeres [10]. With SoC, the deviation can spread to gaze opposite that of the palsied muscle. Absence of A- or V-patterns, or changes of one geographic pattern—say of a V-pattern, to an A-pattern—is a consequence of SoC. The secondary actions of cyclovertical muscles, and even of horizontal rectus muscles, can change from, say esodeviation to exodeviation, from incyclo- to excyclodeviations, or even from hypertropia to hypotropia. The absence of subjective cycloptropia in cyclovertical muscle palsy (mainly oblique palsy) is not only due to cyclofusion and to monococular sensory adaptation with reordering of the spatial response of the retinal elements along new vertical and horizontal retinal meridians, analogous to “abnormal retinal correspondence (ARC)” [11], but it is also the result of the SoC involving muscles with opposite cycloduction. All the factors resulting in spread of the comitance can obscure the diagnosis [12]; they should be kept in the astute clinician’s mind for a correct diagnosis and a proper management.

IV. Motor Disorganization Symptoms

These include vertigo, headaches, confusion and disorientation, mainly due to “past-pointing”.

“Pastpointing” or “false orientation”, is an error of egocentric optical localization of visual objects in their relations to the observer’s body [11]. It is temporary and it usually disappears after few weeks: that is why the symptoms are transient. The egocentric localization will soon become adapted to the new relationship between excessive innervation sent to the palsied muscle and the true localization [11].

V. Binocular Vision:

Suppression and Amblyopia

The most dramatic sensory anomalies that may
develop in the pediatric age group following CNP are suppression and amblyopia. The devastating effects of amblyopia in infants on the visual system, namely on the lateral geniculate nucleus (LGN) and on the visual cortex, were found by Hubel and Wiesel in their experiments on kittens [13]. This was later proven by histologic changes in humans. These changes could be irreversible if amblyopia takes place very early on, and if it lasts longer than a few days. We described a clinical, easy, reproducible and reliable way of determining visual acuity and ruling out amblyopia in infants and preliterate children in a previous publication [14].

VI. Loss of the Binocular Cortical Neurons and Decrease of the Cortical Neurons Responding to the Deprived/Deviated Eye

The neurophysiological experiments of Hubel and Wiesel—later further developed by many investigators [11]—where amblyopia of varying degrees was produced by unilateral lid sutureting or by artificial esotropia in visually-immature rhesus monkeys, and the study of response characteristics of neurons in the striate cortex and pre-striate cortex of these monkeys showed the following: a) only a small number of neurons were binocular, that is responding to stimulation of either eye; b) in animals with severe amblyopia, only a small number of cortical neurons were driven by the deprived/deviated eye; c) with less severe amblyopia, many neurons received input from that deprived/deviated eye; d) a procedure of cross-patching can lead to a total switch-over in ocular dominance, so that now all cells are driven only by the initially-deprived/deviated eye; and e) a judicious choice of timing and duration of cross-patching produced an animal with equal numbers of cortical cells driven by either eye, but in which very few neurons were binocular [15].

VII. Binocular Competition

Before the impressive work of Hubel and Wiesel in the nineteen sixties, the existence of a sensitive period (SP) of visual development was not known. The effects of monocular visual deprivation during the SP on vision and on the visual system, namely the LGN, the visual striate cortex, and the visual association cortex, were not known.

Their first series of experiments involved early monocular visual deprivation in newborn kittens and adult cats. Their results clarified the concept of the SP [13]. The duration of the SP varies among species: in humans it extends between birth—or actually a couple of weeks after birth—and 6, 7, or even up to 9 years of age.

In their later studies in the 1960s, Hubel and Wiesel showed an unexpected result: bilateral ocular occlusion resulted in less severe anatomic and physiologic changes in the LGN and the visual cortex. This result led them to come up with the concept of “binocular competition” and the inhibitory role of the stimulated/fixating eye—in monocular visual deprivation—on the deprived eye [16]. This concept clarified several clinical observations: a) A well-known observation is that in unilateral congenital cataract, the management results are indeed extremely poorer than in bilateral congenital cataract: the same density of cataract, if equal bilaterally or present in an only eye, could have very good management results; b) suppression scotoma is only present under simultaneous binocular stimulation; but then, when one eye is covered, the then strabismic eye exhibited no such scotoma [17].

In CNP, the impact of binocular competition on the visual system (sensory and motor) is devastating and should be seriously taken into consideration and prevented in the preoperative observational period.

VIII. Rotation Deficits: Duction/Version

Failure of duction in the field of action of the palsied muscle can be partial, complete or even absent, since only a part of the total strength of a given ocular muscle is necessary for full ocular movement; therefore a partial weakness may not be seen with duction. If not evident, the deficiency in duction can be evidenced by a) the difficulty in maintaining fixation in the extreme field of gaze of the paretic muscle, b) nystagmoid movement on attempted rotation toward the affected muscle, and c) only noting relative differences between the two eyes during binocular rotations, thus comparing yoke muscle actions. This asymmetry in ocular rotations between the 2 eyes remains the best clinical tool for evaluating versions, with at least 89% of the healthy population having less than 5% difference between maximum versions in all gaze positions tested [18].
IX. Associated Muscle Anomalies

When an EOM becomes paretic, there is a change of the central innervational outflow in all the muscles in both eyes, resulting in secondary innervational deviations. It is important to understand the manifestations that these secondary deviations assume, since they can become so prominent as to obscure the diagnosis, and hence prevent proper management of the original paretic muscle.

In some cases, these deviations can fortunately be temporary and may all disappear when the paresis improves. In other cases, the muscles involved in these purely-innervational deviations, may develop contracture/shortening or chronic stretching/elongation which have to be treated in conjunction with the paretic muscle. In others, the paretic muscle may improve or recover, but the secondarily-contracted muscles remain and perpetuate the deviation, making it necessary to treat them as a separate entity. The etiology of the development of secondary muscle anomalies and secondary deviation is based on Hering’s Law of equal innervation [19] and on Sherrington’s Law of reciprocal innervation [20].

The most commonly encountered secondary muscle anomalies –innervational and/or mechanic-are:

a. Overaction/Contracture of the Direct Antagonist:

When an EOM is put in a “slack” or “loose” condition, as when its antagonist has lost its tonus, it develops contracture. When contractured, it is shortened by loss of sarcomeres. When stretched, its sarcomeres are “higher on the length-tension curve” and will thus pull harder than normal at any given innervation. They become overacting [10].

b. Further Elongation of the Paretic Muscle:

Simultaneous with the ipsilateral sarcomeric loss, there is sarcomeric gain[10] in the paretic muscle with lengthening of that muscle. The sarcomeric gain has the same effect as progression of the paresis, because the paretic muscle now has too many sarcomeres for its normal length, and is “down the length-tension curve” [10].

The reversal of this process can occur when surgery, mainly weakening of the direct antagonist, takes place.

c. Overaction of the Yoke Muscle in the Opposite Eye:

When the paretic eye is made to fix in the field of the paretic muscle, the palsied muscle requires more nervous energy to fulfill its movement than if it were normal. Therefore an excessive innervation is sent, not only to the paretic muscle, but also to its yoke muscle in the opposite eye via Hering’s Law of equal innervation –provided the paralyzed eye is the fixating eye.

Remember that incomitancy in the angle of strabismus in palsy of a muscle is mainly due to 2 factors: a) failure of a palsied muscle to move the eye in the field of its action, and b) overaction of the yoke muscle when the palsied eye fixates.

d. Primary and Secondary Deviations: Early in a course of palsy, primary and secondary deviations are often very well marked: if an eye with a paretic muscle is required to fix in the primary position, or more especially in its field of maximum action, a greater-than-normal innervation to the muscle is required in the central effort to accomplish the required movement. An equal amount of excessive (greater than usual) innervation goes to the yoke muscle according to Hering’s Law. The normal yoke muscle, receiving excessive innervation, moves the eye a greater-than-usual amount and is said to overact. Such an overaction, consequent to Hering’s Law, would only occur if a “fixation duress” in the fixating, palsied eye is exerted. The resulting larger deviation is termed “secondary deviation”.

Note that secondary deviation due to “fixation duress” occurs in two other conditions: a) in co-contraction syndromes, such as in Duane Syndrome, when the affected eye fixates, and b) in severe mechanical restrictions, especially when the restrictions are operative in the primary position and when the eye with restrictions fixates.

A “primary” deviation exists when the eye with normal muscles is required to fix, and the fellow eye with the paretic muscle lags behind. This results because the paretic yoke muscle receives the usual amount of equal innervation from its normal yoke –fixing, controlling eye, but moves the eye less than the usual amount because of paresis. The “primary deviation” (non-paretic eye fixing) will be way less in magnitude than the “secondary deviation” (paretic eye fixing).
This difference between primary and secondary deviations will obviously be greatest in the field of maximum action of the involved muscle, since the differences between overactions and underactions are enhanced in this position. However, there will also be some differences between the primary and secondary deviations in the straight-ahead gaze, but of a smaller degree since the paretic muscle is also acting in the primary position, although to a lesser extent.

This factor of “primary” or “secondary” deviation will create difficulty in measuring deviations. It is best measured by the quantitative prism cover test as it exists under the patient’s habitual condition of fixing: this may be a primary or secondary deviation. However, it is an absolute rarity when a patient fixates with his or her paretic eye. Therefore, measurement of the deviation under the patient’s habitual condition of fixation is almost always measurement of the “primary deviation” — which is the “basic” deviation. So, one should always have the sound fixing eye in paresis, in order to get the “primary deviation”.

Another accurate way to measure the “primary deviation” and avoid the much larger “secondary deviation” is the “deviation-quantitative cover test with prisms” described by Jampolsky in Duane Syndromes [4]: in the forced primary position, the examiner forces fixation with the affected eye with the prisms in front of the affected eye (base-in in exotropia, base-out in esotropia, base-up in hypertropia an base-down in hypotropia) while observing the fellow normal eye under cover. During changes of prisms, the examiner moves the head from right to left to maintain fixation and to easily observe the overshoot, out and in, or up and down, of the fellow, normal eye until there is better prism neutralization. This “fixation duress” exponentially overshoots the fellow, covered eye in or out, up or down, until the straight-ahead position is obtained with the appropriate prisms before the forced, fixing, affected eye. That relieves the “fixation duress” by bringing the image of the object of regard on the fovea of the affected eye. The amount of prisms, used before the affected eye, that straightens the fellow, sound eye determines the actual angle of deviation (Figures 1, 2, and 3, right, next column). This clinical test used by Jampolsky in determining the deviation in Duane Syndromes can be as well used in cases of severe palsy or severe restrictions.

**e. Underaction of the Yoke of the Antagonist:**

This is called the “inhibitional palsy of the contralateral antagonist”, described by Chavasse [7].
Whenever there is paralysis of a muscle, the antagonist in the same eye is able to move the globe in its direction of action with less effort than it normally does, since it lacks the restraining effect of the normal tonus of the paralyzed muscle. The innervation required for the antagonist will be subnormal. A subnormal stimulus is sent, therefore, to the yoke muscle of that antagonist, whenever the paralyzed eye is used to fixate: only when the paretic eye is fixing, and only when it is fixing opposite to the field of action of the paretic muscle, that the inhibitional palsy of Chavasse is elicited in the fellow eye.

A mnemonic rule [21] to identify the muscle involved in an inhibitional palsy in all isolated cyclovertical muscle palsies would be to change oblique to rectus—or rectus to oblique, right eye to left eye—or vice versa, and keep superior or inferior unchanged.

In horizontal rectus muscle palsies, the yoke of the antagonist is the same rectus muscle in the fellow eye: The MR or the LR muscles of the fellow eye in respectively MR and LR muscle palsies.

f. Synergistic Hyper and Synergistic Hypo:

Less well known and less encountered secondary muscle anomalies in cyclovertical muscle palsies are described by Urist [9] and called “synergistic hyper” and “synergistic hypo”. Urist points out the presence of two “synergistic hyper” forces: overaction of the synergist of the antagonist, and overaction of the synergist of the yoke. He also describes two “synergistic hypo” forces: underaction of the synergist of the paretic muscle, and underaction of the synergist of the yoke of the antagonist (Figure 4, right). So, when underaction of the depressors is produced in one eye, there will tend to be underaction of the elevators in the opposite eye, or vice-versa; and conversely, when overaction of the depressors occur in one eye, there will be overaction of the elevators in the opposite eye and vice versa.

Starting with a known paretic vertical muscle [9], by constructing the diagram shown in Figure 4, all of the possible secondary vertical deviations can be easily determined. To explain this, the steps of constructing the diagram are described by Urist [9] and reemphasized in our paper [21] as follows: represent underaction of the muscles by a solid triangle, and overaction of the muscles by arrows pointing in the direction of overaction. Place the base of the triangle in the field of action of the paretic muscle of the involved eye; and reverse this triangle in the contralateral eye.

Taking a right SO palsy as an example, there will be underaction of the right eye looking down, so the triangle is placed with its base downward on this eye. For the left eye, the base is reversed. All muscles are then marked in relation to their fields of action.

g. Secondary Vertical Deviations in Horizontal Muscle Palsies:

Although the respective functions of the LR and MR muscles is to abduct and adduct the eye, effects of palsy suggest they also play a role in the vertical alignment of the eyes [5].

Moreover, in LR palsy, the SO muscle could be recruited to help abducting the affected eye. The secondary depression action of the SO then causes hypotropia (HoT). In MR palsy, the SR muscle could be recruited to help adduction, resulting in hypertropia (HT) due to the SR secondary elevation action.

On the other hand, the loss of fusion in LR or MR palsy may cause disturbance of the vertical tonic vergences [6], resulting in associated HT or HoT, and/or disturbance of the tonic cycloverticyles, resulting in secondary cyclotropia.

Figure 4 (Khawam, Fahed): Adapted from Urist: secondary deviations in right superior oblique muscle palsy.

X. Permanent Muscle Changes

Following innervational overaction or underaction that may develop in all EOMs following a CNP, two courses may take place.

a. They remain innervational, or

b. More often they may develop secondary muscle contractures in which muscle and fascial structural changes occur.

This process is explained as follows [10]: the
increased neural impulses sent to the antagonist of the palsied muscle as well as to all other overacting muscles cause these muscles’ lengths to shorten, with sarcomeres disappearing at the tendon-myofibril junction, thus shortening their anatomic length. The decreased neural impulses to the palsied muscles and all other underacting muscles allow the muscle length of these muscles to increase and to elongate, due to new sarcomeres that appear at the tendon-myofibril junction, thus increasing their anatomic lengths.

We will now consider the different ways available to treat conservatively patients with CNP in their preoperative observational period (see Table). We will also examine the impact of each treatment on the above-cited symptoms and problems faced in CNP.

**B. Table (Khawam, Fahed): Methods of treatment of cranial nerve palsies.**

<table>
<thead>
<tr>
<th>I- Occlusion of the Paretic Eye</th>
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<tbody>
<tr>
<td>II- Occlusion of the Sound Eye</td>
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<td>III- Occlusion of the Sound Eye along with Prisms before the Paretic Eye</td>
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<td>V- Prisms before both the Sound and the Paretic Eyes</td>
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**I. Occlusion of the Paretic Eye**

**a. Advantages:**
- It prevents symptoms of motor disorganization, diplopia, and abnormal head posture, cortical competition, the inhibitory Palsy of Chavasse, and “secondary deviations”.

**b. Disadvantages:** It encourages contracture/overaction of the antagonist of the paretic muscle and further elongation of the palsied muscle (progression of the paralysis). It also encourages amblyopia in the pediatric age group and enhances ocular misalignment. It worsens rotation deficits.

**II. Occlusion of the Sound Eye**

**a. Advantages:**
- It prevents suppression, amblyopia, and diplopia. It stimulates the paretic muscle, therefore preventing its further elongation, and hence preventing contracture of the antagonist muscle via its inhibition by Sherrington’s Law. Finally, it prevents binocular competition.

**b. Disadvantages:**
- It worsens symptoms of motor disorganization, the AHP, and the “secondary deviations”. It encourages the onset of the inhibitory palsy of Chavasse.

**III. Occlusion of the Sound Eye Along with Prisms Before the Paretic Eye**

In addition to the above-listed advantages in occluding the sound eye, the addition of prisms before the affected eye remedies to the disadvantages listed in simply occluding the sound eye: it eliminates symptoms of motor disorganization, the AHP, the “secondary deviations” and the inhibitory palsy of Chavasse by allowing foveation in the affected eye.

**IV. Occlusion of the Paretic Eye Along with Prisms Before the Sound Eye**

It does not solve or prevent any of the above-cited disadvantages of a mere occlusion of the paretic eye.

**V. Prisms Before Both, the Sound and Paretic Eyes**

**a. Lateral Rectus Palsy:**
- Guibor [22,23] in the 1950s for treatment of CNVI palsy, advised a prism method of reducing hypertonicity in the antagonist MR muscle. His goal was to prevent contracture of the antagonist MR muscle with a reasonable expectation that some patients with LR paralysis would recover some muscle function with time. The Guibor concept was as follows: if one could direct the motor apparatus toward the side of the paralyzed LR muscle, then the antagonist MR would be relaxed according to Sherrington’s Law of reciprocal innervation[20].

  Guibor increased gradually the prisms before the sound eye as much as tolerated, with the addition of sufficient base-out prisms before the affected eye, to obtain fusion.

  The concept of Guibor to prevent contracture of the MR in CNVI palsy by placing base-out prisms before the sound eye was generalized and used in different EOM palsies.

**b. Medial Rectus Palsy:**
- Prisms base-in would be used before the fellow eye that direct the field of gaze towards abduction,
thus stimulating the affected MR muscle of the palsied eye and allowing inhibition of the direct antagonist LR muscle therefore preventing its contracture.

Additional base-in prisms are then used over the palsied eye in order to neutralize the deviation and secure fusion.

**c. Superior Rectus/Inferior Oblique Palsy:**
Prisms base-down would be used before the sound eye to displace subjectively the binocular field upward, toward the side of the paralytic eye thus preventing contracture of the affected eye depressors via Sherrington’s Law of reciprocal inhibition. Additional prisms base-up are used before the paralytic eye to neutralize the deviation.

**d. Inferior Rectus/Superior Oblique Palsy:**
Prisms base-up would be used before the sound eye in order to prevent –by the same reasoning-overaction/contracture of the affected eye elevators, the SR or the IO muscles. Additional prisms base-down will be used before the paretic eye to neutralize the deviation.

**e. Third Nerve Palsy:**
The concept of Guibor to stimulate the paretic muscle and hence preventing contracture of the direct antagonist muscle by virtue of Sherrington’s Law of reciprocal inhibition has no place in third nerve palsy because of the multiple EOMs involved.

The Guibor prism method appears sound in theory, but with obvious practical difficulties; expenses of such a method could be high. We believe occlusion of the sound eye fulfills all the expectations of prisms before the sound eye in terms of directing the field of gaze toward the palsied muscle, thus preventing contracture of the direct antagonist of the paretic muscle, and preventing further elongation of that muscle.

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**C. Our Way of (Protocol for) Managing CNP in the Acute Phase and During the Observational Period**

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**I. In the Pediatric Age Group**

Notice that symptoms of motor disorganization are usually absent in the pediatric age group.

In this category of patients, symptoms of motor disorganization usually lack, but two main concerns should be taken into consideration regarding the under-developed/immature visual system:

1) Amblyopia
2) Binocular competition and its devastating effects a) on the binocularity: loss of the binocular cortical neurons and marked decrease of the cortical neurons responding to the amblyopic eye, and b) on the sensory visual system: an indirect effect resulting in more severe loss of vision and more severe organic lesions and physiologic disorganization of the LGN and visual cortex.

**a. Patients with Fusion:**
Fusion is usually due to an abnormal compensatory head posture. We believe, despite the presence of a single binocular vision, that alternate occlusion of the sound eye is beneficial. It prevents contracture of the paretic muscle’s direct antagonist and prevents further elongation of the paretic muscle, on occlusion days. It allows fusion and the integrity of the binocular cortical neurons on the days of no-occlusion. A second option in this category of patients is to do nothing.

**b. Patients without Fusion:**
We advise alternate occlusion of the sound eye every other day to avoid amblyopia, further elongation of the paretic muscle, and contracture of the paretic muscle antagonist. We try, as soon as possible, to restore fusion with the use of prisms split before both eyes.

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**II. In Adults**

The main concern in adult patients with CNP are the permanent muscle changes that develop following innervational involvement of most of the EOMs in both eyes, followed by “muscle length adaptation” and permanent mechanical muscle changes.

**a. Patients with Fusion:**
One option is to do nothing but observe the patient, even if fusion is achieved by an AHP.

If the patient is annoyed by the head turn or chin elevation/chin depression posture, prisms, base-directed toward the side of the head malposition are
used in order to shift the head to an “erect position”, and direct the field of gaze further away from the palsied muscle (Kestenbaum-like shifting of the malpositioned head to the primary position and directing the null zone further away towards its eccentric position [24] in congenital nystagmus).

In muscle palsy with fusion—as well as with congenital nystagmus with eccentric “null zone”—with head turn or chin-up/chin-down position, there develops an abnormal proprioception with an abnormal correlation between ocular, labyrinthine, and neck reflexes resulting in a visual attitude different from that of “normal subjects”, that is, the head erect and the eyes looking straight ahead. In the condition of abnormal proprioception, there is an abnormal relation between the head malposition and the field of gaze (angle α in Figure 5, right ->). By placing prisms base-directed towards the eccentric head posture, the abnormal relation between AHP and eyes remains the same; however, both eyes and head move together in the same direction—keeping the same anomalous relation—away from the head malposition or away from the palsied muscle, directing the field of gaze opposite to the affected muscle—and in CN, away from the malpositioned head- and moving the head, in the same direction and with the same abnormal relation toward an “erect” position. The shift of both head and eyes is equal to the power of the prisms of equal strength placed before both eyes [25] (Figures 5 and 6, right ->).

b. Patients without Fusion:

We start immediately with occlusion of the sound eye with the purpose to direct gaze towards the paretic muscle, thus preventing its further elongation (gain of sarcomeres) and simultaneously preventing contracture of its direct antagonist (loss of sarcomeres) via Sherrington’s Law of inhibition.

If the patient is annoyed with severe symptoms of motor disorganization in the first few days after the onset of CNP, occlusion of the paretic eye can be done—on this unusual and occasional circumstance—only for a few, limited number of days, until past-pointing symptoms disappear. If symptoms last longer, then prisms before the paretic eye are used, along with occlusion of the sound eye, to relieve the patient.

Then, as soon as possible, prisms are used before both eyes, enough to restore fusion.

Figure 5 (TOP) & 6 (BOTTOM) (Khawam, Fahed): See text, prior column. Right LR palsy. Head turn to the right and fusion. TOP: Eyes in primary position. Abnormal “relation” (angle α) between the eyes and the abnormal head posture (face turn). BOTTOM: With prisms base/right of equal strength placed before both eyes: both eyes and head turn to the left, keeping the same abnormal “relation” (angle α): The head is now “erect” or "straight".

Our other concern, in adults with CNP (if we cannot restore fusion with prisms) is disturbance of the tonic vergences: the horizontal, the vertical, or the incyclo-/excyclo-vergences resulting, respectively, in secondary “sensory” horizontal, vertical, or torsional deviations. Occlusion prevents these “sensory deviations” to develop, since the beneficial effect of (complete) occlusion (no stimulus) are multiple: a) it
avoids the irreparable diffusion amblyopia in the pediatric age group; b) eliminates the binocular competition; c) prevents misalignment by eliminating the negative factor of input differences and therefore improves motor coordination, hence preventing secondary/associated deviations.

We personally believe prisms placed before the sound/fixating eye do not achieve more than simple occlusion of the eye as far as shifting the field of gaze toward the paretic muscle in order to prevent its further elongation/deterioration and to prevent contracture of its direct antagonist. We only use prisms before the sound eye when the total amount of prisms split before the two eyes is tolerated and is able to restore fusion.

In the presence of cyclo torsion associated with vertical deviation, the total amount of prisms that can neutralize the vertical deviation will simultaneously correct the cyclo tertia, especially if the latter is of moderate amount, that is not exceeding 10 or 12 degrees.

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Ocular Disorders of the Mona Lisa (Strabismus) and Other Famous Paintings in the Louvre, Paris

JAMES L. MIMS III, M.D.

ABSTRACT: The History of Medicine and Medical Specialties such as Ophthalmology, and the Subspecialty of Strabology extend back to the beginning of recorded history. Before there was photography to record physical abnormalities of living creatures, there was art and artists who did the recording in their works. Fortunately, many such recordings have been preserved, usually in museums, such as the Louvre, in Paris, France. They provide the graphic evidence of the same medical problems which our ancestors suffered and survived which we still do but now understand and can ameliorate better.

The purpose of this paper is to provide several examples of these art works, to illuminate these historical aspects of medicine.

INTRODUCTION

Every year, more than 8 million visitors enjoy the most visited and respected art museum in the world, the Louvre in Paris, France. This past Spring, I had the privilege of being among those 8 million. The most famous of all the paintings in the Louvre, the Mona Lisa (Figure 1, next page), is one of ten known paintings attributed to Leonardo da Vinci, and one of the five displayed in the permanent collection of the Louvre. The painting is displayed behind dark, smoky bullet-proof glass, and, with the added impediment of the large crowd of tightly packed tourists always in front of it, is best appreciated in reproduction. Indeed, her obvious ocular pathology is only really appreciated by study of a good reproduction [This confirms an opinion we recently read elsewhere that going to the Louvre to see the Mona is sort of useless. It is said, because of the security measures they provide, 'tis better and more rewarding just to look at a good reproduction somewhere..., so just turn the page and look at the author's copy and that is probably as near as you are ever going to get to the real thing. -Ed]

The Mona Lisa

Traditional legends regarding the Mona Lisa abound, and are probably mostly not true. It is probably reliable that Leonardo started work on a portrait of Lisa Gherardini, wife of the merchant Francesco del Giocondo (hence the work’s Italian name, La Gioconda) in Florence around 1503. He took it with him, unfinished, when he migrated to France at the behest of King Francois I just prior to 1518. The spurious suggestions that the work is “an idealized female figure of the human conscience”, an idealized portrait of the Virgin Mary (The Virgin of the Rock by da Vinci is surely enough.), or even a self-portrait are all proven to be false and fanciful ideas by recognition of the Mona Lisa’s ocular pathologies.
Twenty years ago, the American Academy of Ophthalmology made much of the discovery that a preliminary drawing of La Gioconda revealed that in the oculomotor primary position she had an obvious esotropia. For many years, a large framed poster produced by the AAO of that picture of Mona Lisa has graced a wall in my office, (Figure 2, below, left) and in the poster she is looking into right gaze. Leonardo’s passion for realistic depiction is well known from his notebooks, so it is probable that she was as orthotropic as he has portrayed her as she looks into left gaze in Figure 1. Obviously, she had esotropic Duane’s Syndrome of the right eye, but he portrayed her looking left, her pathology-free most flattering direction of gaze!!

Two other probable ocular pathologies are seen in her eyelids and peri-orbital area. (Figure 3, below). The bulge in both her right lower lids are probably chalazia, The lump on the left side of her nose just above the medial canthus is probably a verruca vulgaris, a common wart! (Hardly an idealized depiction of womanhood, and since da Vinci was not known to have a wart on the left side of his nose, the Mona Lisa is probably not secretly a self-portrait!

Figure 3 (Mims III): Mona Lisa, close-up view of bulging lower eyelids (chalazia?) and verruca vulgaris on the left side of her nose.
Georges de La Tour’s The Cheat with the Ace of Diamonds

Fast forward to the Baroque painters influenced by Carvaggio. Around 1633-1639 Georges de La Tour painted one of his most famous paintings, “The Cheat” (Figure 4, above). This is actually an allegory. The innocent young nobleman on the right is about to be seduced by what was regarded as among the three worst social sins of the 17th century, gambling, wine and sexual indulgence. The young woman with the turban is proffering wine, the shifty eyed cheat has an extra card behind his back, and the figure in the center is supposed to be a harlot, who with her eyes and her hand gesture is urging her accomplices to act. Her eyes are directed up and to the right, and other ophthalmologists have been tempted to think she has overaction of the left inferior oblique. I spent a total of at least 30 minutes studying this painting on two occasions, and I have concluded that her ocular gaze version (up-right gaze) is probably normal (Figure 5, below). This dilemma does illustrate that in some superior oblique palsies there may be a tendency to over-diagnose overaction of the ipsilateral inferior oblique, when the most important pathology that can be improved by strabismus surgery may, in fact, be contracture of the ipsilateral superior rectus, as suggested clinically by finding hypertropias across the lower field.
**Figure 6 (Mims III): François I, King of France (no obvious ocular pathology) painting by Jean Clouet, Paris, 1485-1540**

The final painting in this brief report is by an unknown French artist who was heavily influenced by the really high quality artists brought to France in 1516 by François Premier (Figure 6, above). François I was the King of France who, by bringing the finest Italian painters of the day (Leonardo da Vinci and Raphael most prominently) to continue their work in France, transformed French painting from a provincial primitive style to an “Italianate” style of which this painting is a great example. The artist is unknown but the date, 1566, is known. Flautist with One Eye will be missed by the casual tourist rushing through the Louvre; I spent a total of four full days in the Louvre this Spring and found this painting in a small, darkened room adjacent to Figure 6. To see the Flautist, you have to press a button to turn on a light that momentarily illuminates the painting. (Perhaps they didn’t want to scare small children.) This painting (Figure 7, below) is not reproduced in compendia purporting to be complete such as *The Louvre: All the Paintings*. Obviously, even though it has been a part of the permanent collection for centuries.

**More to Enjoy (References)**

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LEFT-SIDED PREDOMINANCE IN PSEUDOESOTROPIA

NADIA HESHAM, M.D., JOHN W. SIMON, M.D. and JITKA ZOBAL-RATNER, M.D.

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ABSTRACT: Pseudostrabismus, or more precisely, pseudoesotropia, is commonly encountered in pediatric ophthalmology. In our practice, the left eye was reported by parents as being deviated more frequently than the right eye. (p = 0.001). We attribute this laterality to the fact that most parents (92%) are right handed. They therefore tend to hold their children with their left arm, and to feed them with their right hands, in both cases viewing the child’s left eye in the adducted position.

Received for consideration October 10, 2011; accepted for publication November 2, 2011. Correspondence: Dr. Nadia Hesham, Department of Ophthalmology, Lions Eye Institute, 1220 New Scotland Road, Suite 202, Albany, New York 12159. Email: HeshamN@mail.amc.edu

INTRODUCTION

As neither eye is actually strabismic in pseudoesotropia, the suspected laterality of the condition has never been investigated (1-3). Over the course of several years, we have noticed that, in cases where the family is able to designate a deviating eye, it is more often the left. We undertook a chart review to determine whether our observation is indeed accurate and, if possible, to explain the phenomenon.

METHODS

This study, approved by the Albany Medical Center Institutional Review Board as an exempt study, conformed to the requirements of the U.S. Health Insurance Portability and Accountability Act. An electronic search was conducted using billing records for patients from the practices of the second and third authors from January 2005 - September 2009 with the diagnosis of pseudostrabismus (ICD-9 code 92004). Patients were excluded if, on chart review, a true strabismus was apparent. Patients were also excluded if amblyopia or structural eye pathology was suspected after examination. Patients who returned for follow-up and found to have a true strabismus or other eye pathology were likewise excluded.

RESULTS

Records from 932 patients were identified as eligible for review. Parents had been asked which eye they suspected was deviating. In 323 cases (36%), no specific eye
was identified. In 235 (26%), the right eye was suspected and, in 363 (38%), the left eye was suspected. The left eye was identified more often than the right (p = 0.001) via t-test statistical analysis.

**DISCUSSION**

In our practice, we show parents the Pseudostrabismus brochure developed by the American Academy of Ophthalmology (4). One illustration (**Figure**) shows a child with pseudoesotropia looking slightly to her left, with the nasal sclera in the right eye covered by the epicanthal fold. Parents often respond that their child’s eyes look “just like that, only the problem is in the left eye”.

We have often speculated about the explanation for this apparent left-sided predominance in pseudoesotropia. Most parents (92%) are right-handed (5). As a result, they are apt to carry their child on the left hip and to offer a bottle, or other food, using their right hand. In either case, they tend to view the child’s left eye in the adducted position. We believe parents see that the left eye appears to cross more often than the right simply as a result of the child looking to the right on such occasions.

**REFERENCES**


**Figure (Hesham, Simon, Zobal-Ratner):**

*Pseudoesotropia with the apparent deviation in the right eye.*

*(Courtesy American Academy of Ophthalmology)*

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ABSTRACT: A 26- year-old woman who had a left head tilt since childhood was undergoing left inferior rectus resection to correct her left inferior rectus paresis. During the surgery, when the inferior rectus was isolated and engaged with an eye muscle hook, the muscle tore (snapped) completely into two pieces, 8 mm posterior to insertion. Fortunately, we were able to find the proximal portion of the muscle and, after a 3 mm resection, of the distal yet attached 8 mm portion, the proximal and distal portions were sutured together with a non-absorbable suture.

After nine months followup there was significant diplopia, and the preoperative left head tilt and left hypertropia remained, so a left inferior rectus re-resection was done. At the end of 18 months followup after the second procedure there was no binocular deviation (strabismus) in primary position nor in any other gaze positions, but there was a mild ocular motility infraduction deficit present on left and down gaze.

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INTRODUCTION

Loss of a rectus muscle either during or after eye muscle surgery for strabismus is one of the most serious complications of strabismus surgery. If it is not treated, a lost muscle will lead to consecutive strabismus, diplopia and large limitation of eye movement in the direction of the lost muscle (1,2,3). A snapped or torn muscle is one in which the insertion to the globe remains intact but the muscle belly has been completely ruptured
across its width at some point posterior to the muscle insertion (4). Extraocular muscle snapping can occur during either strabismus, vitreoretinal surgery or after trauma.

Few cases of “snapped” or broken muscle have been reported in the literature. The overall incidence for snapped muscle was estimated to be 1 in 5000 surgeries (5).

In this case report we describe how we found the proximal end of the snapped inferior rectus muscle and, because of significant diplopia and residual hypertropia 9 months after the surgery, with re-resection of the inferior rectus, we were able to completely resolve the binocular deviation.

CASE REPORT

A 26-year-old woman with left head tilt since childhood was referred to our clinic. Past medical history was negative and she could not remember trauma. In ophthalmic examination, best corrected visual acuity was 20/20 in both eyes. Refraction was OD: -4.00 -0.25×180 and OS: -3.00-0.75×15.

Ocular motor examination revealed a left hypertropia (LHT) of 6 prism diopters (PD) in primary position and 12 PD LHT in left and down gaze (see Table 1).

Table 1: Patient’s preop’ deviometry.

<table>
<thead>
<tr>
<th>PD</th>
<th>LHT 4Δ</th>
<th>XT 10Δ</th>
<th>LHT 6Δ</th>
<th>XT 12Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>6Δ</td>
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<td>10Δ</td>
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<tr>
<td>12Δ</td>
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The remainder of the ophthalmic examination was normal.

According to patient’s deviometry our assessment was an isolated left inferior rectus (IR) paresis; therefore, IR resection was considered for treatment. In the operating room, under general anesthesia, forcedduction testing was negative. After limbal conjunctival incision, Tenon’s capsule and intermuscular septum was dissected. The IR muscle was hooked and the eye was rotated upward without excessive force. Suddenly, the IR tore completely in two at a point then measured 8mm from its insertion to the globe.

Figure 1 (Akbari et al): Distal part of snapped inferior rectus muscle. Hematoma on the margin of the muscle is noticeable.

Immediately, we performed a meticulous posterior exploration and we found the torn end of the proximal part of the IR muscle.

Because of corrugation of the distal muscle edge at the tear, nearly 3 mm of distal muscle was resected and the distal and proximal portions of the IR muscle were sutured together with a non-absorbable wire, 5-0 mersilene suture.
One day after surgery, her oculomotor examination showed 18 PD LHT in primary position and 26 PD LHT in left and down gaze (Table 2, below):

Deviometry, day after 1st surgery.

There was still a prominent left head tilt and the patient now complained of diplopia. We therefore prescribed a Fresnel prism in front of both eyes to reduce diplopia. After one week followup the patient had 12 PD LHT in primary position, a reduction in the deviation from one day postoperatively.

Two weeks after surgery, because of the reduction in the deviation, the prism in front of the right eye was deleted. After six weeks, the deviation size had not changed.

At nine months followup, due to permanent diplopia and significant head tilt, we considered and planned a second stage of surgery for her deviation. (Table 3, below).

Deviometry, 9 months after 1st surgery, before 2nd surgery.

After conjunctival incision, the IR was found at site of normal insertion (6 mm from limbus) and the previous non-absorbable suture was visible. A conventional 4mm re-resection of the IR at the insertion was done.

After this second surgery, her diplopia was resolved in all gazes and there was no detectable deviation in primary position.

After another 18 months of followup, no binocular deviation was detected in primary position and only a mild infraduction deficit was present in left and down gaze (see Figure 2, directly below).
DISCUSSION

Loss of a rectus muscle can occur as a complication of strabismus surgery, trauma, orbital or retinal detachment surgery (4,6,7). A snapped or torn muscle is one in which the insertion to the globe remains intact but the muscle belly has been completely ruptured across its width at some point posterior to the muscle insertion on the globe (4).

One retrospective study demonstrated that half of snapped muscles are the medial rectus and the next most frequent muscle is the IR. The overall incidence of snapped muscles is estimated to be 1/5000 surgeries (5).

The IR has two insertions: two-thirds of its length from its origin, the IR divides into a capsulopalpebral head (CPH) and an ocular head. The CPH divides into two layers surrounding and fusing with the sheath of the inferior oblique. This joining forms the central portion of Lockwood’s ligament (8,9).

The facial IR strands anterior to Lockwood’s ligament are called “the capsulopalpebral fascia”. The superior portion of the capsulopalpebral fascia proceeds anteriorly and ends at the limbus. It is called anterior Tenon’s Capsule. The inferior portion of the fascia sends strands through the orbital fat to the orbital septum. The anterior portion of this fascia with tarsal muscle fiber ends at the tarsus of the lower eyelid (8,9).

When, at surgery, the eyeball is retracted superiorly with a strabismus hook under the inferior rectus, the eyeball is rotated much more than the lower eyelid. Since the capsulopalpebral system is firmly attached to the orbital septum, skin, tarsus and the periosteum of the orbital floor the eye ball, cannot move freely as it is somewhat tethered by the capsulopalpebral head of the IR. These two opposite forces may contribute to tearing of the IR muscle at the point between the IR insertion and the origin of CPH (8,9).

The IR muscle is often reported to pull apart with mild traction and this has also been referred to as the "pulled in two syndrome" (6). This may be more common with presumed extra ocular muscle pathology, previous damage from trauma and/or surgery, in elderly or debilitated patients (7). Because the muscle rupture typically occurs well posterior to the ocular head of the muscle insertion into the sclera, retrieval of the proximal, lost aspect of the muscle is rendered more difficult and is often impractical.

Many reports have shown that after a lost muscle occurred, the proximal part could not be identified and reattached. So, the surgeon has to use other methods for keeping the eye monocularly straight ahead in the primary position as well as binocularly aligned with the other eye (1,2,3,6,10).

Kowal et al. (6) reported 3 patients with snapped IR during strabismus surgery, and did not find a proximal part. They performed a modified inverse Knapp procedure (6). There are also a few reports, about inferior oblique anterior transposition to compensate for a snapped but unrepaired IR, each with different results (1,2,3).

Fortunately, we found the proximal portion of the snapped IR muscle at the time of surgery. But because of significant diplopia and the residual head tilt, we found a difficult choice about the second stage surgical plan.
We had two options: the first option for surgery was ipsilateral antagonist superior rectus recession with a posterior fixation (myopexy) suture of contralateral IR. This method has been recommended by other authors (4,7).

The second option was performing the same procedure as our first operation on this patient (for any patient with IR palsy).

Although, the first option seems more safe, we, however, chose the second option for surgery. One week after IR re-resection the patient had no diplopia, no head tilt and no tropia detectable in primary position. At the end of another 18 months followup, a mild infrafractional deficit was present on left and down gaze; but, no strabismus was detected in primary position. The patient has no diplopia and no abnormal head posture.

To our best knowledge, this is the first published case of a snapped IR that was treated with two stages of IR resection with a final acceptable result. This report demonstrates that, repetitive surgery (re-resection) on a snapped and retrieved muscle is possible and this approach itself can lead to an acceptable result. In fact, after IR snapping, we were able to resolve the problem without using another extra ocular muscle.

REFERENCES

PAUL E. ROMANO, M.D., M.S. Ophthalmology

Research originally performed at the Department of Ophthalmology, University of Florida College of Medicine, Gainesville, Florida. See Editorial for publication rationale.

ABSTRACT: Techniques for the adjustment of surgery intraoperatively (especially those termed Stage I and II techniques) have proven maximally successful in improving surgical results for comitant strabismus. Stage III adjustments (end-operative) have been described but not studied.

In a retrospective study of 20 eye muscle procedures in 12 patients with neuroparalytic and mechanical strabismus, the usefulness of various intraoperative adjustment techniques stage I, II, and III was investigated for the first time.

Stage I adjustments (adjusting the surgical plan based on the binocular misalignment following induction) were not helpful. Stage II adjustments (R. Bedrossian’s technique: adjusting the amount of surgery performed to create an actual change in binocular alignment under anesthesia matching the change in alignment desired clinically) were appropriate for horizontal mechanical and (all) vertical cases but not appropriate for horizontal neuroparalytic cases.

Stage III adjustments, at the end of surgery, were appropriate in virtually all cases (20 muscles, 12 patients). Significant overcorrection, well beyond the theoretically ideal final intraoperative binocular alignment of 30 PD (prism diopters) was appropriate in all cases, but varied with type of case: Verticals (all) required a 5-10PD overcorrection. Horizontal mechanical cases required a 22-30 PD overcorrection. Horizontal neuroparalytic cases required a 15-38 PD overcorrection, in the last group, in each case, graded according to the presence of contractures and the size of the preoperative deviation.

The use of Stage III (and Stage II as noted above) adjustments brought postoperative binocular alignment to orthotropia +/- 10 PD in all cases, the conventional standard for satisfactory results in strabismus surgery.

Presented at the VI International Strabismological Association meeting, Queensland, Australia and recorded in the Transactions for that meeting.

Correspondence: Email: perxbvg@colorado.net. Author has no financial interest.
INTRODUCTION

Few or no guidelines exist for quantitating or guiding eye muscle surgery specifically for neuroparalytic or mechanical and restrictive forms of binocular misalignments, incomitant strabismus, as do for comitant deviations.

In such situations, the strabismus surgeon may find him- or herself in this position confronting such a problem without prior warning and is personally even if he had references in the operating suite, yet totally unprepared.

There is a correlation between the binocular alignment under general anesthesia, and the binocular alignment clinically when awake (1,2,3). This correlation is $A=0.8P+30$ where $A$ is the binocular alignment under general Anesthesia and $P$ is the Preoperative binocular alignment, both in prism diopters (PD). (Sign convention is esodeviations are negative, exodeviations are positive). Strabismic and all patients are significantly more divergent under general anesthesia than awake on the order of magnitude of 30 PD.

The possibility then exists of measuring the binocular alignment at the end of eye muscle surgery, while the patient is still under general anesthesia, and adjusting the amount of recession or resection being performed, so as to place the eyes in that particular alignment which should produce the desired binocular alignment (orthotropia) postoperatively. (That binocular alignment under anesthesia is 30 PD exotropia by the Apt Test for comitant strabismus.) (1).

We call this end-of-the-procedure action a Stage III adjustment. Stage I adjustments, in contrast, are based on the binocular alignment immediately after general anesthesia induction. If this binocular alignment is anomalous, the planned surgery is adjusted. A Stage II adjustment, as described by Bedrossian in 1983 (4) is adjusting the extraocular muscle recession sutures at the end of surgery so that the amount of binocular alignment change intraoperatively from the surgery in PDs is that same amount of PDs of correction which was desired clinically preoperatively.

We have used Stage I adjustments (2,3,5,6) to significantly improve our success rate from comitant strabismus surgery to the very best rates reported. Bedrossian (4) has had similar success with Stage II. No one to our knowledge has here-to-fore used or reported Stage III intraoperative adjustments. The purpose of this paper is to report our experience with these Stage III intraoperative adjustments.

METHODS

In both a retrospective and prospective manner, we studied the results of 20 eye muscle procedures in 12 patients with neuroparalytic and/or mechanical strabismus which we had performed at our institution since we had started using intraoperative adjustments.

At first, standard and augmented standard amounts of surgery for comitant strabismus were used for these special cases. We noted that in spite of good binocular alignment under general anesthesia at the end of these cases undercorrections regularly resulted. Therefore, based on the amount of undercorrection observed in similar cases, surgery was increased using adjustable sutures (intraoperatively at the end of the procedure) to produce an appropriately overcorrected anesthetized binocular
alignment at the end of the case (Stage III adjustment).

RESULTS (See Table)

Stage III intraoperative adjustment proved very useful in obtaining a good postoperative binocular alignment in cases of horizontal and vertical neuroparalytic and mechanical strabismus.

The best final binocular alignment under general anesthesia for these case was not, however, the same as that for cases of comitant strabismus (exotropia 30 prism dipters or 2 mm by corneal light reflection test; i.e., Apt or modified Apt Test). Rather, a very significant overcorrection was required (See Table).

The amount of overcorrection indicated was 5 to 10 PD for (all) verticals; 22 to 30 PD overcorrection for mechanical horizontal strabismus; and 15 to 38 PD graded overcorrection for neuroparalytic horizontal strabismus. The horizontal overcorrections must be ADDED TO OR SUBTRACTED FROM THE 30 PD EXOTROPIA “ideal” Stage III final binocular alignment.

Stage I adjustment, altering the amount of surgery based on the alignment of the eyes following induction of anesthesia, was also studied and found to be NOT useful in these cases.

However, Bedrossian’s 1983 method (Stage II intraoperative adjustment) (4) was, similar to Stage III adjustment, also a VERY GOOD guide to intraoperative adjustment of surgery in the horizontal mechanical cases, and a useful guide in vertical cases.

Stage III adjustments (overcorrections as outlined above, and in the Table) produced satisfactory binocular alignment (OT +/- 10 PD) both vertically and horizontally in all cases, except for one horizontal deviation in a case of thyroid myopathy s/p orbital decompression but she was a success by Stage II adjustment.

DISCUSSION of RESULTS

We are unaware of any reports of this specific type of treatment of these cases of complex strabismus, either published before this report was given nor since then. Bedrossian’s related work and reports on Stage II adjustments have been cited in the introduction and were found (see Table) to provide some similar guidance to that we have reported for Stage III adjustments in both vertical neuroparalytic and mechanical cases, and horizontal mechanical cases.

CONCLUSIONS

In the absence of finite reported or published guidelines for eye muscle surgery for neuroparalytic and mechanical strabismus, satisfactory postoperative binocular alignment can best be achieved if, at the end of surgery under general anesthesia, the surgeon measures the binocular alignment and adjusts the surgery performed to produce the desired overcorrected binocular alignment (Stage III adjustment). (Adjustable suture techniques which are reported to be used postoperatively may facilitate this but our published studies do not support such techniques per se.)

Vertical neuroparalytic and mechanical strabismus should be overcorrected 5 to 10 PD...
TABLE: Intraoperative Adjustment of Eye Muscle Surgery for Incomitant Strabismus, Results and Recommendations from 20 Procedures, Stage I, II, and III.

<table>
<thead>
<tr>
<th>Type of Strabismus</th>
<th>Intraoperative Adjustment Technique</th>
<th>Application of Stage III adjustment in O.R. measured by Apt or modApt Test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HORIZ NEUROPARALYTIC</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ex: VI N 2' MVA</td>
<td>no</td>
<td>OVER RX</td>
</tr>
<tr>
<td>VI N 2' BT surgery</td>
<td>help</td>
<td>15-38 PD</td>
</tr>
<tr>
<td>III N 2' BT surgery</td>
<td>help</td>
<td>GRADED BY FORCED DUCT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MAGN OF DEV (1.0-2.5mm CLRT)</td>
</tr>
<tr>
<td><strong>HORIZONTAL MECHANICAL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ex: old MR trauma</td>
<td>no</td>
<td>VERY GOOD</td>
</tr>
<tr>
<td>old slipped MR</td>
<td>help</td>
<td>OVER RX</td>
</tr>
<tr>
<td>old lost MR</td>
<td>help</td>
<td>22-30 PD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.5-2.0mm CLRT)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>do not over rx</td>
</tr>
<tr>
<td></td>
<td></td>
<td>fresh surg trauma</td>
</tr>
<tr>
<td>ex: IV N 2' plagiocephaly</td>
<td>no</td>
<td>GOOD</td>
</tr>
<tr>
<td>radiation scarring</td>
<td>help</td>
<td>OVER RX</td>
</tr>
<tr>
<td>blow out fracture</td>
<td></td>
<td>5-10 PD</td>
</tr>
<tr>
<td>thyroid myopathy</td>
<td></td>
<td>(0.3-.7mm CLRT)</td>
</tr>
<tr>
<td><strong>VERTICALS (all)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ex: IV N 2' plagiocephaly</td>
<td></td>
<td>GOOD</td>
</tr>
<tr>
<td>radiation scarring</td>
<td>help</td>
<td>OVER RX</td>
</tr>
<tr>
<td>blow out fracture</td>
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<td>5-10 PD</td>
</tr>
<tr>
<td>thyroid myopathy</td>
<td></td>
<td>(0.3-.7mm CLRT)</td>
</tr>
</tbody>
</table>

Legend: see text for definition and description of the 3 stages and details of technique; abbreviations: roman numerals for cranial nerve paralyses, MVA: Motor Vehicle Accident brain trauma; BT: Brain tumor; MR: Medial Rectus extraocular muscle; CLRT: Corneal Light Reflection Test, i.e. Hirschberg. ET: Esotropia; XT: Exotropia; OT: Orthotropia; HT: Hypertropia; hT: hypotropia; PD: Prism Diopters; Rx: Treat

by corneal light reflection tests. Horizontal mechanical cases should be overcorrected 22 to 30 PD and horizontal neuroparalytic cases 15 to 38 PD (overcorrected with reference to the standard 30 PD exotropic binocular alignment [by corneal light reflection test] which corresponds to orthotropia postopera-

Stage II adjustment is also appropriate in vertical and horizontal mechanical cases. Stage I adjustment should not be used here but is best for comitant strabismus. (Stage III adjustment cannot be recommended at the present time for comitant cases of strabismus).
however. Our observations to date suggest there are exceptions which we do not yet understand in this situation. Stage III does, however, work very well for the incomitant types of cases presented here.)

Please note this final caution: All these recommendations and all the measurements of binocular alignment by corneal light reflection test or other method under general anesthesia are accurate only if the eye muscles are totally paralyzed.

At the time these surgeries and studies were originally carried out, this required very deep inhalation anesthesia which most anesthesiologists did not like to use, with good reason, at the beginning and end of surgery. This was however just when it is needed for these techniques. The best alternative at that time were non-depolarizing muscle relaxants such as Pancuronium or Atrocurium. After adjustments were accomplished, these could be reversed without prolonging the duration of the general anesthetic agents used at that time.

Those general anesthesia drugs have been replaced with safer agents such as propofol (7) but we note that our original intraoperative adjustment techniques can and are being utilized currently under the new general anesthesia without any significant changes.

REFERENCES

Statistics

Eyes or Subjects: Are Ophthalmic Randomized Controlled Trial: Properly Designed and Analyzed? Lee CF, Cheng ACO. Fong DYT. Ophthalmology January 2012. [Authors Conclusions]

There is currently substantial heterogeneity in the quality among published ophthalmic RCTs in terms of proper use of study design, sample size calculation, randomization method, and statistical tools. Future ophthalmic researchers are suggested to consult a statistician and to follow some guidelines such as CONSORT statement when performing an RCT to improve further the quality of the clinical trial. (Chun Fan Lee, PhD, 31 Biopolis Way Nanos 02-01, Singapore 138669, Republic of Singapore)

(clipped for those of you who swear by randomized controlled study data and refuse to recognize anything that isn’t. Your Gods have feet of CLAY. See our numerous prior editorials and citations regarding your Unscientific addiction-per)

Vision / Visual Acuity / Amblyopia


The dichoptic based perceptual learning therapy employed in the current study improved both monocular VA of the amblyopic eye and stereo function, verifying the feasibility of a binocular approach in the treatment of childhood amblyopia. (Email: pamela.knox@gcu.ac.uk)

Binocular Vision

Binocular Fusion of Luminance, Color, Motion and Flicker - Two Eyes are Worse than One. Anstis S, Rogers B. Vision Research 2012; 53:40-46. [Science Direct Highlights]

● Each eye saw random spots that were spatially identical but differently colored, fused in a stereoscope.

● Left eye saw a cross of yellow spots embedded in green spots, right eye saw same yellow cross but embedded in red spots.

● When fused binocularly, all spots looked yellow and the cross disappeared.

● Monocular colors could be retrieved by a special afterimage technique.

● Study shows what information was conserved, discarded and/or reached conscious awareness.

Pseudophakic Monovision May Offer Better Intermediate Vision than Multifocal IOLS. [from the AAO Academy Express]

Researchers prospectively compared visual function and patient satisfaction (see new significance in Hyde Park editorial, page 66, later in this issue -per) in 21 patients implanted with bilateral diffractive multifocal IOLS (AcrySof ReSTORE SN6OD3) and 22 patients with monofocal IOL monovision (AcrySof SN60WF). Visual exam at three months showed bilateral uncorrected distance vision and near vision were slightly better in the multifocal group, but the difference was not statistically different. Monovision patients had significantly better intermediate vision and less difficulty using computers without glasses. Monovision patients also had a slightly higher overall satisfaction score and significantly fewer
complaints of glare and halos. *(J Cataract Refractive Surg, March 2011)*

**Strabismus Pathophysiology**


*(Dr. Ronit Gilad, Dept Neurology, Edith Wolfson Medical Center, Holon 58, Israel. Fax: 972-3-502-8681)*

**Elderly Strabology**

**Convergence Insufficiency Significantly Affects Parkinson’s Patients’ Quality of Life.** Ophthalmology, January 2012. [from the AAOs Academy Express]

This prospective, case-control study assessed whether dopaminergic medication alone or in combinations with deep brain stimulation improved visual and ocular motor function in Parkinson’s patients during the symptomatic “on state”. Additionally, it evaluated patients’ vision-related quality of life. Results showed Parkinson’s patients suffer from substantial convergence insufficiency compared to controls, which fluctuates throughout the day in association with the dosing schedule of medications, which complicates ophthalmic management and adversely affects vision-related quality of life.

**Extraocular Muscles**


Sustained IGF-1 treatment of both MR muscles in infant monkeys results in bilateral increases in myofiber size and nerve density in treated MR and in the antagonist but untreated LR. As alignment is maintained, central adaptive mechanisms must control compensatory changes in the yoked muscles. *(Dr. McLoon. Email: mcloo001@umn.edu)*


Anterior segment optical coherence tomography (AS-OCT) can image the structure of horizontal rectus muscles well and provide good reliability and accuracy in measurement of the limbus-insertion distance. *(Xinhai Ye, dryxh@189.cn)*

**Stereopsis / 3D**

**Egocentric Localization**

**Environmental Surfaces and the Compression of Perceived Visual Space.** Bian Z, Anderson GJ. J Vision ARVO 2011. [Authors conclusions, edited]

...the perceived visual space when viewing a ground surface is less compressed than the perceived visual space when viewing a ceiling surface and that the perceived layout of a surface varies as a function of the type of the surface. *(Anderson@ucr.edu)*

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.
HYDE PARK EDITORIAL: The Editor’s Soapbox, Sandbox & B’LOG
(Prehistoric) Since 1985

CONSCIOUSNESS: A Function of Stereoscopic Vision via the Egocenter.

Stereoscopic 3 Dimensional depth perception vision remains the Acme, Epitome and GOAL of all of both (monocular) vision and Binocular Vision. It’s the Very Foundation of Ego-Centric Localization, which is Your Very Own Cornerstone of SPACE and your respective unique world around you, and us, each live in 3D.

In the last issue of last year, the last before this one, we featured egocentric localization in both the lead Editorial and here in Hyde Park. We repeat the Headline title and subtitle above. Here’s a leftover tad from what we said then here: “First, we think we are right about egocentric localization,... [and] have looked a bit further about it. The egocenter has been anatomically located [in the hippocampus, connected to the posterior parietal and occipital lobes]. It gets input from all senses, some of which have been traced to it. It may be the very site of our sense of CONSCIOUSNESS!!!! It is probably organized like a topographical map of the external world (which naturally would be 3D and based on stereoscopic visual information and experience).

“DO WE NEED STILL MORE REASONS FOR THE IMPORTANCE TO LIFE OF NORMAL BINOCULAR VISION AND STEREOPSIS? WHAT MORE IS THERE?
“Excuse me but I have some more very heavy reading to do!...”

Which I did to some extent more since then, essentially confirming what I said above and herewith confirm. So it really is “where we live” literally and figuratively. We in the eye business are literally also a bit behind the times in recognizing the relative immense importance of the egocenter and binocular vision to our very being and to life, and to its unbelievable sophistication and complexity, combining and integrating all the neurological input which is continuously received and processed, and most of which is immediately re-accessible to our consciousness when needed or desired. No wonder that sleep and rest is so essential to sanity. That is some motherboard under our O.S.!

And everything in our eye business is literally “in service” to it.

From all this reading we are happy to confirm our long held quote motto-logo that has appeared on our Table of Contents each issue for the last decade, peculiarly appropriate now for our more recent current enthusiasms for stereoscopic depth perception binocular vision and the egocenter where it goes:

“... the belief that one’s view of reality is the only reality is the most dangerous of all delusions...”-Watzlawick, 1976

Especially now that we understand that the “reality” out there is a function of our egocenters, and these cannot be identical for thousands of reasons, no? - even though we know it is only one real physical world out there around us for us to sense with all our senses... everyone sees it differently, right? Or as the egocentrists say, there is no single fixed POV in the egocentric world (Point of View). You can pick any one from infinitely numbered POVs in your own egocenter.

“Conscience” and religion have been very much in the political news lately thanks to the Obamacare cancer. Wonder where they hang out?

re 3D movies: Mims IV sent a clipping which noted these are against nature in that: “the viewer’s eyes [have to] focus on the screen while converging on the constantly changing distance of the 3-D images”... well, yes and no. “30% of viewers become nauseous...” I missed them when I went... maybe 4D... ?:

PEARLS BEFORE SWINE

By Stephen Pastis
Do we really need all those ordinary cameras anymore? All smartphones have cameras, many two. Some do 3D too, just like this ad, some not yet. And they all have 8 MP memories which is more than enough for video and even huge enlargements. They are starting to have many other elements of today’s remaining “real” cameras... even Kodak quit. I may have bot my last recently, a Samsung very wide angle 21mm but 14X to a decent telephoto of 300 mm. I still have a couple of great 20x30" posters on the wall of local scenes I took a decade ago on a 2 MP ! point and shoot camera.

3D views and viewing are everywhere, on the next page to enhance endoscopic cancer detection... using OCT (optical coherence tomography) to go beneath the surface ... microscopically...
3-D OCT delves deep for improved cancer screening

CAMBRIDGE, Mass. — A new optical coherence tomography system can look below the surface with greater detail than traditional screening methods can, enabling high-speed 3-D imaging of microscopic precancerous changes in the esophagus or colon.

Endoscopy is the current method for screening esophageal and colon cancer, which are diagnosed in more than 1.5 million people worldwide each year, according to the American Cancer Society. However, standard endoscopy examines only the surface of tissues, so malignant changes occurring at greater depths may be missed.

OCT, however, examines layers of tissue below the surface and can visualize structures just a few microns in size. Over the past two decades, OCT has become commonplace in ophthalmology, where it has been used to generate images of the retina and to diagnose and monitor diseases such as glaucoma. It can be performed inside the body through the use of miniature fiber optic scanning catheters or probes, either on their own or in combination with standard endoscopes, colonoscopes or laparoscopes.

"OCT for cancer imaging is one of the most challenging research applications because of its clinical complexity and the demands on the technology," said OCT pioneer James Fujimoto, professor of electrical engineering at MIT. "We have been working on endoscopic OCT imaging for over a decade. We believe that this is one of the most important current areas of investigation and has a high potential for future impact."

The new endoscopic OCT imaging system from MIT works at record speeds to capture data at a rate of 980 fps (equivalent to 480,000 axial scans) — nearly 10 times faster than previous devices — while imaging microscopic features less than 8 µm in size. At such high speeds and resolution, the system promises to enable 3-D microscopic imaging of precancerous changes in the esophagus and colon.

The findings were described in the July issue of Biomedical Optics Express (doi: 10.1364/BOE.2.002438).

In collaboration with clinicians at the VA Boston Healthcare System and Harvard Medical School, the team has begun to investigate endoscopic OCT as a method for guiding excisional biopsy — the removal of tissue for histological examination — to reduce false negative rates and to improve diagnostic sensitivity.

The device has been used in animal models and in samples of human colons that have been removed during surgical procedures. Further development and testing of the technology is needed before it can be used in humans. The team expects to introduce the next-generation OCT technology within the next half-year, Fujimoto said.
Do It Yourself 3D anything Prototyping

Getting Started

© The biggest hurdle for would-be 3D-modelers isn’t the price or the complexity of the software—it’s the overabundance of options. There are pro-level modeling and rendering suites, finicky engineering tools, and simplified-to-the-point-of-uselessness art apps. In search of an entry point, I found an app called Tinkercad.

This free application runs inside a Web browser on nearly any PC or Mac and contains just the right level of functionality—it’s capable enough for real 3D modeling, but not so complex as to put you off. It’s a solid-modeling program—much like most professional CAD apps—which means that its models are an agglomeration of points in space rather than a hollow group of stitched-together polygons. With its emphasis on solid, volumetric materials, this type of modeling is particularly well-suited for 3D printing, and Tinkercad has a button that creates a 3D-printer-ready file instantly.

To get started, navigate to tinkercad.com and create a free user account. I was presented with a blank slate—or, in the parlance of 3D modeling, an open work plane. Building in Tinkercad is conceptually simple: In the Add mode, you select a shape—a box, a pyramid, a cone, or a cylinder—along with a size. You then stamp this shape into 3D space; clicking and dragging will stretch the shape as far as you want. The Sub (for “subtract”) mode lets you use the same shapes for object removal. One of the easiest practical projects is a shirt button: With the Add tool, stamp a disc that’s 16 mm wide and 2 mm thick. With the Sub tool set at 3 mm wide and 2 mm thick, stamp out two buttonholes near the center of the disc. That’s it.

My first nonbutton project was admittedly a modest one: a Popular Mechanics paperweight. It was to be about 4 inches wide, with a thick, 1-inch-deep base. Our trademark PM lettering would be perched on top. (Swap for your initials if you want to follow along.)

Modeling a shirt button takes about 2 minutes; my paperweight took a great deal longer—about an hour, including 15 minutes to get used to the app and no small amount of trial and error. Getting used to the stamp-and-cut behavior of the program was the biggest challenge; in its current incarnation, there’s no way to move or resize an object—a block, for example—after it’s been placed on the grid. Getting the spacing of the letters right took a few tries.

The core of the paperweight was composed of just seven shapes: one block for the base, four blocks for the M, and one block and a disc for the P. After creating the core, it
was a matter of cutting, trimming, and adding accents. I subtracted small pieces of material from the middle of the M to match our iconic typography and carved out a hole for the P. I used small cubes to stamp serifs onto the letters and did cleanup with a 1 x 1-mm subtract tool. The end result was good enough. The next step was to make the paperweight real.

3D Printing
© Popular Mechanics has devoted a fair number of pages to a company called MakerBot, whose 3D printers can create small, plastic prototypes in just a few minutes—all they need is a healthy supply of ABS plastic and a 3D-model template from a program like Tinkercad. MakerBot’s machines are affordable, but only relatively: At $1000, they’re far cheaper than industrial prototyping machines, but out of reach for most hobbyists—including me. Plus, I was making a paperweight, and MakerBots print only in plastic. I wanted something with heft—glass or stone or even metal. That’s where Shapeways, an on-demand 3D printing service, came in.

Sending my model to Shapeways was a two-step process: From Tinkercad I exported my project as an STL file, the industry standard for 3D printing; at shapeways.com I just clicked the Upload button on the front page. Ten minutes later, I got a message saying that my model had been approved, and I was presented with nearly 20 choices of materials. For $25, I could have the project printed in ceramic. For $290, I could have it cast in sterling silver. I made my selection (sandstone, $36) and I would have my paperweight within two weeks. I’ve already chosen a followup project: Ever the picky tech editor, I’m custom-designing a case for my smartphone.

Advanced Prototyping
© Software for 3D modeling ranges in price from free to thousands of dollars and varies hugely in complexity. Tinkercad is a fantastic starting point, but you’ll hear a lot of other names when you dive into the world of 3D modeling.

SketchUp, by Google, is a popular program by virtue of its tie-in with the company’s 3D-mapping program, Google Earth. (Google encourages users to populate its virtual maps with 3D replicas of real buildings.) Blender, another free program, is an open-source alternative to professional programs such as 3ds Max. It’s versatile—there’s really nothing you can’t do in Blender—but it has a steep learning curve.

The app 123D, new from Autodesk, the company that makes the industry-standard professional CAD software, is a well-balanced tool kit for intermediate 3D-modelers and connects natively with Shapeways for easy 3D printing. Once you’re comfortable in a program like Tinkercad, you can find your way in 123D.

There’s room to grow with Shapeways as well. Once you’re satisfied with your design, you can list it for sale on the site. You choose the material options and markup, and they handle the ordering, printing, and shipping. “Three-D modeling started as a hobby,” says van der Linden, but after launching a product line that includes kinetic toys, geometric sculpture, jewelry, and desk lamps (Shapeways can print objects up to about 27 x 15 x 22 inches), it’s quickly becoming a healthy source of income.

Roman Vasylyev, a freelance designer, had been building model cars and airplanes for years before discovering 3D printing. His obsessively detailed World War I-era aircraft models now net a steady stream of income. (The top seller is a 1:44 scale model of the Caudron G.4, a French biplane bomber.) “I was really surprised that 3D modeling has become another branch of my hobby,” he says, “and now, my work.”

Modeling and printing in 3D has everyday applications, too. In an hour or two, Tinkercad or 123D can help you replace that once-irreplaceable knob on your priceless old guitar amp, for example. Last year a man named Duann Scott asked the manufacturer of his high-end baby stroller, Bugaboo, for a part to repair a broken hub lock. When the company said it would charge $250, he scoffed. Scott took apart the hub, figured out what he needed, modeled the parts on his own, and had them printed in stainless steel. Total cost: $15.
And 3D helps us with our eye research using OCT and elsewhere: Here, “CTH” is a family of software codes developed by the Sandia National Laboratories for multidimensional shock physics analysis (1993).


Methods: ...studied with abattoir-fresh porcine eyes... CTH ... produce[d] robust geometric and constitutive models of eye and orbit, providing a comparative 3-D finite volume model [of]... ballistic[s].

Conclusions: ... at least two trajectory-dependent mechanisms for optic nerve rupture... Tangential glancing blows produce strain-rate rotation avulsion, abscising the optic nerve [from the globe...] with minimal... Internal disruption, whereas off-center direct impact produces slower rotation-rebound evulsion, traumatizing the globe and breaching the nerve posteriorly... (expected...more commonly ... likely... clinically masked by ... intraocular injury). (Dr. Sponsel. Email: sponsel@earthlink.net) [edbolded]


This method provides measurable 3D images of the LC which enable comprehensive evaluation of the LC configuration. This technique should facilitate the investigation of the LC in glaucomatous eyes. (Dr. Tae-Woo Kim. twkim@snu.ac.kr)

Stereopsis / 3D
Egocentric Localization

...the perceived visual space when viewing a ground surface is less compressed than the perceived visual space when viewing a ceiling surface and that the perceived layout of a surface varies as a function of the type of the surface. (Anderson@ucr.edu)
h2. **strabology**: we (actually our patients really) still have to suffer from these ancient horrible and erroneous prejudices but their presence in the comic strip provide uncontrovertible evidence how they do persist. ![I shall protest] - and motivate you strabologists to eliminate the defect completely.

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h3. **Other ophthalmology**: (non-strabology).......

**Including Vision and Seeing and Looking:**

Do note that this Lip reading is only a very short phase of infant development lasting only 2 to 4 months, beginning of the second 6months of life.

A good few years back at Children’s in Chicago a co-worker, a speech therapist, asked us if we knew what sort of vision it took to lipread. We found nothing in our library so we did a little research and published it. (Romano PE, Berlow S.: Lipreading Performance as Related to Visual Acuity. *Am J Ophthalmol* 1973; 75:136 and . Vision Requirements for Lip Reading. *Am Ann Deaf* 1974; 119:383)

It took only 20/100 (or better vision) when *speechreading* (proper term). So doing this at this age is no guarantee of fully normal 20/20 vision. But determining if a child is in fact lip reading at any time isn’t going to be easy!

From the *American Medical News* January 24, 2012

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**Babies appear to lip read as part of language acquisition.**

*NBC Nightly News* (1/16, story 8, 0:30, Williams) reported, “There’s interesting new research out about how infants learn to talk. While they tend gaze directly into the eyes of those holding them until the age of six months, they often then switch to reading lips, watching mouth movements intently as a way of learning how to sound out words themselves.”

The *AP* (1/16) reported, “Babes don’t learn to talk just from hearing sounds. New research suggests they’re lip-readers too,” according to a study published this week in the Proceedings of the National Academy of Sciences. “Florida scientists discovered that staring around age six months, babies begin shifting from the intent eye gaze of early infancy to studying mouths when people talk to them.”

“Scientists from Florida Atlantic University studied 89 infants ranging in age from four months to 12 months old,” *ABC News* (1/17, Conley) reports. “They also studied 21 adults. Participants watched a 50-second video of a woman reciting a monologue in their native English, while researchers used an eye tracker to determine where they directed their pupils while watching and listening to the video.”

*MSNBC* (1/17, Raymond) points out, “Results showed that at four months of age, babies focused almost solely on the women’s eyes.” However, “by six to eight months of age, when the infants entered the so-called ‘babbling’ stage of language acquisition and reached a milestone of cognitive development in which they can direct their attention to things they find interesting, their focus shifted to the women’s mouths. They continue to lip read until about 10 months of age, a point when they finally begin mastering the basic features of their native language. At this point, infants also begin to shift their attention back to the eyes.”

*HealthDay* (1/17, Preidt) explains, “The finding challenges the conventional belief that infants learn to talk only by listening to people around them, according to the Florida Atlantic University researchers. They also said their discovery may suggest new ways to diagnose autism spectrum disorders.” That is because “contrary to typically developing children, infants who are as yet undiagnosed but who are at risk for autism may continue to focus on the mouth of a native-language talker at 12 months of age and beyond.”
Video on the Brain
TAKING STRIDES TOWARD UNDERSTANDING HOW THE BRAIN PROCESSES STIMULI TO RECOGNIZE IMAGES, RESEARCHERS FIGURE OUT HOW TO PROJECT NEURAL ACTIVITY ONTO A TV SCREEN.
BY STEVE ROUSSEAU

Images are transmitted to the brain via each eye’s optic nerve. A These signals are first processed in the primary visual cortex. B Scientists examine the primary visual cortex to produce images from brain activity.

HOW DO THEY DO IT?
UC Berkeley professor Jack Gallant and his team use MRI to track blood-flow changes in a subject’s primary visual cortex—the brain’s largest visual processing center—as he or she watches a movie. The researchers then create a model of the visual cortex that matches the blood-flow pattern with the images the subject is viewing. Algorithms are applied to compare the brain signals with a catalog of about 5000 hours of YouTube videos. The images that most accurately correspond to the brain activity are compiled into a composite video, which resembles the YouTube footage.

“I think it’s very impressive that they can get these admittedly crude re-creations of our internal representations of video,” says Marcel Just, director of the Carnegie Mellon University Center for Cognitive Brain Imaging, who was not involved in the study.

WHAT’S IT GOOD FOR?
Such brain–visual linkages could one day aid communication with stroke or coma patients. Gallant says that after his team’s technique is refined, it could also record and play back dreams. The obstacle to this is understanding how the brain’s visual processing changes when a person is sleeping or awake. Gallant is confident this will happen. “It’s only a matter of time,” he says.
Ancient, isn’t it. Now expensively updated with unbelievable gadgetry and all those new electronics: touch screens and strobo LCD glasses (3D leftovers?) And professional consultants. It works?

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**TECHNOLOGY**

**Visions of Perfection**

A new training tool has athletes sporting specs to reach their peak performance

In 2008, his third year in the NFL, Packers receiver Greg Jennings dropped a whopping eight passes, tied for seventh most in the league and putting him in the company of such notorious butterfingers as Braylon Edwards and Terrell Owens. But those days are long gone. Jennings has brought that number down in each of the past three seasons, and in 2011 he caught only three that got away, an improvement he attributes in part to a new form of sensory performance training that helps him hone his focus, reaction time and decision making.

The system, developed by Nike’s training division, begins with a visit to a touch-screen kiosk (essentially a hyperintense video game unit, used above by the Lions’ Ndamukong Suh) that tests and tracks visual and sensory performance skills, from depth perception to reaction time.

That information then provides the framework for a custom-designed on-field training program, as each piece of sensory data defines an individual player’s deficiencies and training needs. “We want to quantify everything,” explains Paul Winsper, a Nike performance director. “Every step and every drop of sweat.”

The sweating that starts in the kiosk moves to the practice field, where players don plastic eyewear with liquid-crystal display lenses, dubbed Vapor Strobes, that flash microscopic electric charges, blocking vision in 100-millisecond patterns. As players run drills, the lenses flicker between clear and opaque, the distractions forcing each athlete to anticipate what is coming and focus on his task, whether it be a running back finding an open hole, a quarterback spotting a free receiver or a wideout guiding the ball into his hands.

A recent Duke University study found that the stroboscopic training improves motion detection and central attention, helping boost visual perception. By taking away partial vision with eight levels of obstruction and varying speeds and modes, the strobes build muscle memory—essentially a high-tech football version of a weighted bat.

In effect, “by giving them less information, we force athletes to utilize what we give them more efficiently,” says Dr. Alan Reichow, Nike’s lead sensory performance researcher. “[It’s] essentially stress training on the sensory system.”

For Jennings, who uses the strobes pre- and postpractice in a medley of ball-catching drills, the training has sharpened his in-game focus dramatically. “You can see the ball a little better and react quicker,” he says.

Jennings has even persuaded the rest of Green Bay’s receiving corps to don the eyewear for workouts. The result? In the 2011 regular season the unit’s frequency of dropped catchable balls stood at 7.8%, down from 10.9%—second worst in the NFL—in Jennings’s ‘06 rookie season. And really, who’s to argue with a Packers receiver this year? —Tim Newcomb
The NFL’s Best-Looking Team

In a Turbulent Season That Defies Prediction, We Decide to Rank Each Team by ‘Facial Symmetry’

BY REED ALBERGOTTI

Trying to predict the outcome of the NFL season has always been one of the great fool’s errands in sports. This year, however, it’s just plain foolish.

A summer labor squabble, which led to a vast reshuffling of personnel and a truncated off-season, has knocked the whole league off its axis. Rookie who just got their playbooks are about to be ordered onto the field to execute plays.

So rather than trying to predict the season’s outcome, why not focus on another sort of ranking? Since the NFL’s opening game between Green Bay and New Orleans Thursday coincides perfectly with the start of Fashion Week here in New York, we’ve decided to explore a vaguely related question: Which NFL team is the handsomest?

At the Journal’s request, researchers at Ursinus College in Pennsylvania analyzed the facial structure of a sampling of 520 NFL starters (five offensive and five defensive players from each team). They also threw in two of the most photographed personalities on any team, the owner and the head coach.

To measure each subject’s attractiveness, the researchers analyzed their photograph with a computer program that measures facial symmetry, or how closely the two halves of a person’s face match up. Research suggests this is a reliable measure of how attractive a person is perceived to be. It also suggests that looks are a fair predictor of a person’s career success. A 1994 study found that more-attractive "symmetrical" people are paid about 5% more than the average person while more homely "asymmetrical" people earn up to 10% less.

To be clear, we’re not saying there’s any proven connection between winning football teams and good looks. If anything, it may be an inverse relationship: Teams with particularly attractive players might be guilty of favoring people who are more
Work It, Boys!

Here are the NFL’s 32 teams, ranked by one common measure of attractiveness:

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HISTORICAL NFL FRANCHISES
1. 1992 Dallas Cowboys (97.7); 2. 1975 Pittsburgh Steelers (97.2); 3. 1989 San Francisco 49ers (95.5); 4. 1985 Chicago Bears (95.3); 5. 1972 Miami Dolphins (95.0)

Source: Ursinus College researchers Jennifer VanGilder, Thad Gregory, Rebecca Kamn, Chris Burcham, Dan Horowitz, and Kristen Wampole

VanGilder, a professor of business economics at Ursinus who oversaw the research, said the lure of hiring pretty faces is unconscious. “People are drawn to more symmetrical objects.”

So who wins the NFL’s beauty pageant?

The most attractive sample of players, with an average facial symmetry rating of 99.47, was one of the worst teams in the league: the Buffalo Bills. Their record last season was a woeful 4-12. The least attractive team was the Kansas City Chiefs, a group that won its division last season and made it to the playoffs.

The striking thing about the research, though, isn’t the difference in attractiveness between teams. It’s the fact that the vast majority of NFL players rank 10% higher in facial symmetry than the average Joe. They also seem to be better looking than other athletes. VanGilder also measured the NBA’s All-Star team and found it was less attractive than our sample of NFL players.

Another surprising finding: NFL quarterbacks aren’t the most attractive players. According to this sample, the most attractive position in the NFL is the kicker. The symmetry score for kickers beat the score for quarterbacks by .16%.

The least attractive position of the ones we studied was wide receiver. But with a score of 97.04, these men were still vastly more attractive than average people (who score in the high 80s), VanGilder said.

Even NFL owners and head coaches are extremely good-looking. In fact, as a group, they are more attractive than our sampling of defensive ends and linebackers, safeties, cornerbacks, running backs and wide receivers. Facial symmetry does not change with age, which is why 39-year-old Buffalo Bills head coach Chan Gailey is more attractive than many players. “My wife will be shocked,” said Mr. Gailey, through a team spokesman.

All this may be good news for the NFL, which has embraced outlets like social media, which give players more of a public profile, and has seen its players make star turns on shows like HBO’s “Hard Knocks” and ABC’s “Dancing With the Stars.” The league has also been reaching out to female fans for years.

A sampling of some of the greatest NFL teams in history shows that the NFL is becoming more attractive. The famously undefeated 1972 Miami Dolphins, the 1975 Pittsburgh Steelers, 1985 Chicago Bears and 1989 San Francisco 49ers would all rank near the bottom compared to current NFL teams. If the 1992 Dallas Cowboys played today, they would only rank No. 24.

Facial symmetry is by no means a perfect gauge of attractiveness. Mark Sanchez, quarterback for the New York Jets, isn’t the most symmetrical player in the NFL, but he seems to get the most magazine spreads.

And in case you’re wondering: New England Patriots quarterback Tom Brady (98.52) is about .5% less attractive than his wife, the Brazilian supermodel Gisele Bundchen (98.99%).
Re last pages: we know that facial symmetry is the key to having and being “good looking”, but why the correlation with athletic superiority? Or is that just yet another small but significant factor when people have to make choices of other people. Universal cosmetic plastic surgery and air brush painting is de rigeur for young females. And men are doing it more too. Maybe it will become a mandated freebie under ultimate Obamacare like contraception?

h4 Medicine elsewhere:

Brain Scans May Suggest [predict] Dyslexia in Pre-Reading Children.

Reuters (1/24, Steenhuysen)... When researchers gave brain scans to 36 preschool children, they found that children with family histories of dyslexia, as well as older children and adults who had dyslexia, showed less brain activity in regions including junctions between the occipital and temporal lobes and the junctions between temporal and parietal lobes....

Health Day (1/24, Mozes) adds, In addition, ... among at-risk pre-reading children there was no evidence of activity increases in key frontal lobe brain regions previously linked to dyslexia... [suggesting] that the brain’s method for trying to compensate for... dyslexia does not appear... until after children begin to read.

h5 Other Medical; practice, careers

Medical practice: Well, the following “discovery” will be no surprise to you. Maybe better clip this and keep it around for when the time will surely come when your patient satisfaction ratings will be thrown in your face as a reason to deny you some incremental Obamacare payment vital to your income stream.... but as the final paragraph below notes, patients who are “highest satisfied”... probably because “they got what they want(ed) “have fewer ED visits” and that we all know is the real and absolutely most critical money saver for the government payer... = Stat # 1 for you.

So you better just give your patients “what[ever] they want”. ? Forget “Outcome”?

AMA Mem... Researchers Use Stem C... 2/14/2...

Higher patient satisfaction may not be linked to better outcomes.

Bloomberg News (2/14, Frier) reports, “Patients are more satisfied when doctors give them what they want, though it's not always the best treatment strategy,” according to a study published in the Archives of Internal Medicine. Researchers found that “healthy outcomes didn’t correlate with satisfaction in a study that surveyed more than 50,000 people nationally from 2000 to 2007.”

The Los Angeles Times (2/14, Healy) “Booster Shots” blog reports, “In their first year of participation, participants were asked to rate how satisfied they were with the care they received from their physicians, as well as some health and biographical questions.” During the second year, participants were asked to detail what medications they had been on in the past year, how often they had sought medical care in a clinic or doctor’s office or emergency department, and whether they had been admitted to a hospital for care.

Investigators “tracked a large subset of the participants for an average of 3 1/2 years to discern whether death rates bore any relationship to patient satisfaction.”

The Sacramento (CA) Bee (2/14, Garza) reports that “patients who were most satisfied had a greater chance of being admitted to the hospital and about 9 percent higher health-care costs. Death rates were also higher, according to the survey.” Researchers found that, “for every 100 people who died in the least satisfied group, about 126 people died in the most satisfied group.”

Medscape (2/14, Johnson) reports, however, that the researchers also found that “patients who report the highest satisfaction with their physicians and other healthcare providers have fewer emergency department (ED) visits compared with those who are less satisfied.”
MONEY TAXES ANNUAL REMINDER

MONEY: AVOID Taxes, It’s O.K.

The avoidance of taxes is the only intellectual pursuit that carries any reward.
—John Maynard Keynes

Income TAXES are due in a few weeks! 1. **GO ROTH !!!**

2. The best new advice I have seen is DON’T OBSERVE and submit your 1040 before the first and imminent April 15 deadline: especially if you have not received all the tax notices you know you need, 1099s, 1099Rs, 5498s, K-1 (very frequently LATE). Extension to the second deadline date, October 15 is virtually automatic without any reasons being required. Yes you do have to submit a request to extend (form 4868) and you do have to pay now whatever your best estimate of tax owed is. But otherwise, if you file on April 15, and then get more info that should be part of your return, you have to REFILE your whole return!!!! and that is much more work and much more dangerous (**refiling a return attracts audits**). And this year and every year, you have still another excuse- all the tax changes STILL in the works right now and some of which could or might be EVEN retroactive to (for) last year.

  If you are due a refund, you can ask for it and get it now, too. But be correct.

3. TAXATION AVOIDANCE: WHY it is OK:

  because a Federal Judge says it is: Judge Learned Hand:

  From *The Wall Street Journal* January 25, 2012, Letter to the Editor from Joseph Horton, Lafayette, LA. “... ridiculous for the media to castigate him [Romney] for paying as low a federal income tax rate as possible. It’s called enlightened self-interest, and no one described it better than Judge Learned Hand:

  ‘Anyone may arrange his affairs so that his taxes shall be as low as possible; he is not bound to choose that pattern which best pays the Treasury. There is not even a patriotic duty to increase one’s taxes. Over and over again the Courts have said that there is nothing sinister in so arranging affairs as to keep taxes as low as possible. Everyone does it, rich and poor alike and all do right, for nobody owes any public duty to pay more than the law demands.’ It’s Congress that makes the tax laws to which Mr. Romney conformed. ...”

  **So if you think the tax laws are bad, CHANGE!** Change Congress by Voting them out for someone better....

  And shut out B.O.s current GUILT TRIP oratory: “sacrifice” (they sure don’t) and “fair share” (= gov’t takes ALL and maybe give you a little back). Our current mess is 100% gov’t XS.
5. And you can do your taxes yourself. It is not that hard, especially with the inexpensive software available for all computers.

Don’t forget, that financial analysis says you can only afford to outsource the job, pay someone else to do your taxes for you if you gross more than $13,900,000 (in 2008): Remember this? Outsourcing? WHAT is your time worth?..

Buffet is rich but WRONG. Rite a check, man!

4. WHY, RATHER, IT IS YOUR CIVIC AND MORAL DUTY AS A CITIZEN TO PAY AS LITTLE TAX AS YOU CAN ???:

Your editor’s PUBLISHED “Opinion”, in response to a local news column written by the paper’s resident Democratic tax-more professional propagandist.

HOW BAD YOUR GOVERNMENT IS:
The democrats/democratic press [idem] is so obsessed with taking more of the people’s money by taxing, that they are today (February 16, PRAISING Rick Santorum for a “win” for beating Mitt Romney with the percentage of his income that he pays taxes on: Santorum beats Romney because he paid 28% of his meager one million dollar income last year, while Romney admitted to paying only 15% of his $12 million income. The facts mean that Mitt produced for the government almost two million dollars last year while Rick produced for the government only a third of one million, is totally lost and ignored.

That is the difference between the democratic brain and the republican brain. The democratic brain is permanently and pathologically myopic.

...
About The rest of life: current young people:

This Letter to the Editor, WSJ was about a young women who had publically attacked an airline for not coddling and babying her and repairing her own mistakes and wrongful planning.: He said she:

Like auto insurance., OUR WHOLE CULTURE AND LIFE HAS BECOME NO FAULT ! Except for the government who has gotten really good at finding fault with everyone who doesn’t work for the government! Everyone who works for the government is now totally immune to fault or blame.

Now here is some really very useful information for a lifetime: About cleaning all your monitor e-readers and smartphones’ SCREENS!!! without scratching them or otherwise destroying your investment! from Popular Mechanics, January 2012 by John Herman. Cleaning Your Gadgets.

“... First things first: Barring a few specialty items, most... you need to clean your electronics are either already in your home [in hand] or available at a ... store. [but SKIP:] ‘Electronics cleaning cloths’ and ‘electronics cleaner’ solutions are rebranded, overpriced variations on conventional household products. Most of your gadgets cleaning can be done with three tools: MICROFIBER chamois or pure cotton cloths, distilled water, and ISOPROPYL alcohol. Cleaning any screen should start with a light, dry wipe down with a microfiber... (It’s best to avoid paper towels, which are more abrasive and prone to dragging particulate dirt across the screen, scratching its coating.)” Then wash, rinse with the fluids, dry.

AT LAST: THE REAL REASONS WHY LIFE IS SO DIFFICULT: MANY OTHER HUMAN BEINGS ARE AWFULLY EVIL:


What Makes Man So Beastly.

“The suffering that humans have inflicted on one another and the various ways in which they have justified it has been a longstanding preoccupation of... Bourke.... when she writes of war... the heart of [it is]: destroyed limbs, violated bodies, smashed lives, and the horror and hideous ecstasy of killing. ..... [This book] focuses on what it means to be INhuman and on how the powerful rationalize their INhumane treatment of the powerless, especially the poor, the enslaved, the stateless, prisoners, women, children - and animals. ...the maltreatment of women ... by an Enlightenment humanism that ‘installed only some humans at the centre of the universe.’ Like theology, humanism is ‘founded on espousing hierarchies of humanity.’ Humanism, she says, ‘disparaged “the woman”, “the subaltern”, and “the non-European” even more than “the animal”... less surprising is the frequent failure of those who empathize with animals to feel as strongly about human beings... Ms. Bourke believes that European humanism, as it developed over the past centuries, inevitably classifies the vast majority of human beings as subhuman. ...” (Mr. Tallis is the author of “Aping Mankind: Neuromania, Darwinitis, and the Misrepresentation of Humanity”)
8.5 Segue-way to section 9. Life+ safety+ autos

Sometimes Dear Abby is so helpful and useful we just have to pass it on. SO here’s a

MOTORISTS IN THE WRONG FIND WAYS TO MAKE THINGS RIGHT

DEAR ABBY: “Mild-Mannered Motorist in Virginia” (Dec. 26) asked you for a hand signal to indicate “I’m sorry” to fellow drivers when he makes mistakes behind the wheel. Not long ago, I made a not-so-serious mistake that angered another driver. When I flashed a peace sign, then moved my mouth in an “I’m sorry,” the person’s frown changed to a smile. We then drove on with pleasant attitudes, and I tried to watch my driving more closely.

The peace sign, of course, is hand closed, forefinger and middle finger up as in a “V.” I think the whole world recognizes a peace sign. It worked for me. - FAITHFUL READER IN ARKANSAS

DEAR FAITHFUL READER: I assumed “Mild-Mannered” that my helpful readers would step forward to offer suggestions for an “I’m sorry” signal. And many, like you, mentioned giving the peace sign. Offering more options, my newspaper readers’ comment:

DEAR ABBY: We New Yorkers have honed silent signals to a fine art. When I’m at fault in traffic or other situations where I can’t apologize verbally, I make eye contact, put my hand to my chest to accuse myself, and put my hands in a prayerful gesture to ask forgiveness. This almost always defuses the situation on the spot. Add a smile and you’ve made a friend as well. - LORNA, IN THE CITY

And at least a light colored if not yellow jacket, the kind that are sold in bicycle shops. Look like a worker. (Carrying a noticeable tool or bundle is OK)

My favorite walking cap has stapled around the crown a 2 inch wide strip of red and white diagonally striped reflective car tape. That gives me 360 degree reflection in low light or the dark 24/7. Use strobe flashers too then.

I actually prefer walking over biking just because I can face the traffic passing me and see how they are driving maybe even make eye contact with the driver and leap out of their way if they lose it. I did just exactly that to save my butt at least once.

DEAR ABBY: I, too, have made boo-boos while driving and wish I could have said “I’m sorry,” but the person is usually too busy shaking a fist and screaming what are obviously obscenities to notice. I like the idea of a standard “sorry” gesture.

How about holding one hand up with your palm toward your face for just a second? (As in “I’m ashamed of what I just did.”) It’s simple and lets you keep your other hand on the wheel. - CAROL IN HOUSTON

DEAR ABBY: Why not use the American Sign Language sign tool for “sorry”? Make a fist with your right hand, palm toward the body and place it over the area of your heart and move it in small circles. Of course, the expression on your face pulls it all together. Sign language is used by many people, and the chance that the person you offended may already be familiar with this sign makes it a great way to convey the emotion. - SIGN USER IN OLD LIME, CONN.

DEAR ABBY: If I think the other driver will be able to see me, the gesture I make after a mistake is an exaggerated, slow smack to my forehead – basically, my own Homer Simpson “Doof!” - IN THE WRONG IN MAINE

DEAR ABBY: “Mild-Mannered Motorist’s” letter reminded me of an incident a few years ago. Driving home from work, I was forced to swerve into an oncoming lane by a car driven by a young woman who was so angry. She didn’t see me right. Fortunately, there was a big, black, low slung Hummer behind me. My car iT was facing away from the Hummer, which made it impossible to even see. Suddenly, out of the corner of my eye, I saw her car take a left turn directly in front of me. The Hummer swerved on the road before me, hit her car and the other car hit the wall. She drove on, I called out and we stopped to check her and make sure she was okay. She was. We both agreed to call the police but the Hummer didn’t wait. I was so thankful, but I was also angry. I called the police and they came and wrote a ticket. It was an accident, not a collision, but it was a near death experience for me and the other driver. We both decided it was an accident and we both agreed to move on.

DEAR ABBY: “If you are walking, swinging your arms burns more calories and makes you look bigger, especially if bent out 15 degrees (like a PENGUIN!).

Pedestrian safety: to get that necessary mile or two of brisk walking in every day we often have to use ordinary streets without sidewalks, thereby competing with auto traffic and the risk of getting run down. And where we live the roads are always slick (from that darned mag chloride if nothing else), and drivers do lose control of their cars for no good reason; more than one pedestrian has been run down and killed on the shoulder.

Do what we have recommended bicyclists and other cyclists do. DRESS TO THREATEN CAR DRIVERS! Look official! Look serious! At least wear a SAFETY VEST

By Abigail Van Biver

White Dear Abby

at www.DearAbby.com
or P.O. Box 69440,
Los Angeles, CA 90069

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Hyde Park Editorial ‘BLog

FIRST Quarter of 2012
Quarterly Simms-romano's ©
A Medical Scientific EYE e-Periodical
Pages 53-72
Now a detour for the Marquis of Q.
Followup: Ortiz and Mayweather again.
more again: Marquis of Queensbury “Protect yourself at all times”

We ran this story and the lower picture in the third issue of last year, 2011, volume 164, number 3, with that lower picture blown up to full page size on the last page of that issue, for effect.

We commented on that pic that Ortiz was defying the Marquis of Queensbury by being defenseless when time was no longer”out” in the ring, noting that his right arm was in a low not defending position, even if it was in front of him a bit.

Then I found another photo (top) which was taken obviously just a fraction of a second earlier and before the lower one we had published. It is placed above the earlier published picture since it is earlier in time, and it shows Ortiz with his arms essentially TOTALLY AT HIS SIDES, totally totally defenseless. In the lower pic he is already unconscious now, knees buckling, falling backwards.

This makes what Mayweather did even more horrible and inexcusable morally, even if the idea was suggested by ? “Allowed” or permitted by prediction? By the Marquis of Queensbury’s warning not to do it !!!! (?should=could=would?)

Maybe Manny P. Will repay him for Ortiz if and when they ever meet. For Mayweather’s lady ex too:
News late 2011: Las Vegas: judge sentenced Floyd M. to 90 days in jail after he pleaded guilty to a reduced battery domestic violence charge and no contest to two harassment charges...

He had tangled with his ex-girlfriend while two of their children watched...
X Hard Turn for Auto Makers: Cars Aren’t a Must for Kids.
“... young buyers: Many of their potential customers couldn’t care less about owning a car in the first place. .. They are staying home, using social networks to connect with friends and moving to big cities where mass transit makes car ownership optional. Young buyers want cars that are safe, affordable, compatible with the latest tech gadgetry and good for the environment. Their suggestions helped design the exterior of the two Chevy’s, which GM says look and drive like sports cars but cost around $20,000. GM says it will rely on input from young people to flesh out the interiors. ... draw inspiration from muscle cars and Italian sportsters. They are four-seaters that get 40 miles a gallon, GM North American chief Mark Reuss said. ‘They look confident, expensive and fast.’ ...

The decline in teenage drivers has been underway for year. ... ‘Millennials have more options and more decisions to make than any of us ever had.’ ... ‘A lot of the time students can’t afford to drive: it’s not the car, it’s the insurance and the maintenance,’ ...

Below: Biker safety: BAD biker
A Final word from the editor.

With this first issue for our 27th year we have expanded the size of the journal in number of pages, by half of an old printer’s 4x4 “book” of 16 pages, so it is 72 pages instead of the 64s of recent years. We are also going to try to print the few hard copies we provide to several libraries, etc., in house, to reduce those printing expenses hopefully.

We also have printed below the essentials of our official history but we did not include in this issue the detailed total history as we usually do in this first annual issue in order to meet our contributors’ needs for space, which we are happy to do.

The full history we did is in the first issue for last year, where we left out Hyde Park Editorial to provide space for it. This year, Hyde Park won out.

Hope to see everyone in San Antonio. Have a safe trip.