ESSAY

CAN MIKE MAY BENEFIT FROM OPTOMETRIC VISION THERAPY & REHABILITATION?

(Or, Vision: What is it Good For?)

Leonard J. Press, O.D.

Editor's Note: Dr. Press is a prolific writer and perceptive clinical mind in the discipline of behavioral optometry. It was therefore with great pleasure that I received this essay from him concerning "sight" regained. I trust that you will find this narrative essay as interesting and thought provoking as did I.

Can Mike May benefit from optometric vision therapy and rehabilitation? Or, vision, what is it good for? These may seem like odd questions, but before attempting to answer them, one should know more about Mike May. Blinded at age three by corneas scarred in a chemical explosion, Mike defied odds by breaking world records in downhill speed skiing. Further, he functioned quite well in a world without vision.

He was 46 years old on February 12, 1999, when the former CIA operative and budding entrepreneur accompanied his wife on a chance visit to an optometrist. Jennifer's contact lenses had been bothering her while in San Francisco, where Mike was receiving an award for mentoring the blind. Dr. Mike Carson is an optometrist who practices in San Francisco with Dr. Dan Goodman, an ophthalmologist and corneal specialist.

After Dr. Carson examined Jennifer, he learned that it had been 10 years since Mike had seen an eye doctor. He offered to take a look and optimistically summoned his colleague, Dr. Goodman. After a five minute exam, Mike was informed that, pending a clean B-scan ultrasound, he might be able to regain vision in one eye through a corneal stem cell transplant. As this news was quite unexpected, and would require a radical departure from his current way of functioning, Mike wisely gave the offer much thought before agreeing.

Mike decided that despite the risks associated with the surgery and the systemic medication required to counteract stem cell rejection, the allure of sight restoration was too intriguing to disregard. He forged ahead with the operation. Within minutes of removing the bandage from his eye, it was clear that the operation was technically a success. Yet, it had not been possible to envision the outcome.

To the surgeon it appeared that Mike's pristine optical hardware should have allowed him to see well enough to obtain a driver's license. The problem, though, was that Mike's visual brain wasn't properly programmed to process the visual information he received through his eye.

Blind-sight is a phenomenon where an individual appears to see when vision is thought to be impossible. Mike was the inverse. He exhibited a type of "mind-blindness" where he could not interpret things he was expected to see. Even after six weeks, he was working very hard to make sense of the visual world. He admitted to his wife, Jennifer, that he needed to process every little thing consciously in an effort to understand what he was seeing. Imagine trying to learn a foreign language with an alphabet you can hear, but can't decipher.

This paradox fascinated Ione Fine, Ph.D., an experimental psychologist at the University of California at San Diego. At the time that she discovered Mike, Dr. Fine was on faculty in the Department of Psychology at the University of Washington. The department boasted a rich tradition in childhood visual development, most notably through Professor Emeritus Davida Teller. Fine knew that the rate that babies learn to understand the world suggests that some aspects of vision are inborn and others are learned. Mike therefore provided a unique opportunity for vision scientists to obtain a window into the fragments of vision that babies learn to integrate in an apparently seamless fashion. How would 43 years of total visual deprivation affect the visual perception of a cortex that was presumably normal until age 3? Dr. Fine collaborated with researchers at the Salk Institute and Stanford. In 2003 they published the results of their extensive electrodiagnostic and psychophysical investigations.[1]

Discover magazine had introduced Mike May to the public in 2002 through an article entitled "Sight Unseen."[2] Mike's story has been updated in a special issue of *Discover*, "The Brain," that was published this fall.[3] In addition, the details of Mike's experiences are recounted in a marvelous book by Robert Kurson, entitled: *Crashing Through: The Extraordinary True Story of the Man who Dared to See*.[4] An excerpt from this book, and a *National Public Radio* interview with the author is also available.[5] Kurson poses a number of penetrating questions that provide the optometrist food for thought:

1. Why did Mike chose not to read about his predecessors in history, all of whom seemed to suffer a profound de-

pression for having dared to see after a lifetime of blindness?

2. How would one describe Mike's new vision? Is it what he and the scientists expected? How is his sight different from traditional sight, and what challenges does his new vision pose?
3. There came a time when Mike's struggle with his new vision became so difficult that he nearly destroyed his (corneal stem cell) antirejection medication. Why didn't he simply let his vision go and return to his very full and satisfying life as a blind person?
4. The book often stresses that vision is dependent on knowledge. How is that possible? What are the implications for Mike's new vision?
5. Dr. Ione Fine must teach Mike to do a lot of "cognitive heavy lifting" in order to make sense of what he sees. What is meant by this? How does Mike teach himself to see?

My Introduction to Dr. Fine

Dr. Fine was conversing with Dennis Levi in a Sarasota hotel during the annual meeting of the Vision Sciences Society, when we met briefly. Following the meeting, I sent her this e-mail:

Dear Ione,

I had the pleasure of meeting you briefly in the hallway during the VSS meeting, while you were chatting with Dennis Levi.

As luck would have it, while shopping the lovely St. Armand's Circle in Sarasota the next day, I popped in to the Circle Bookstore and lo and behold Robert Kurson's new book featuring Mike May was on display. I'm sure Kurson took some authoritarian liberties, but I very much enjoyed his depiction of the interaction you had (and evidently still have) with Mike as researcher-collaborator-friend.

Dennis, Sue Barry and I then had the chance to sit down and discuss vision therapy in the context of perceptual learning. That is a subject for another day, but in reading Kurson's account of Mike's personalization of vision (beyond the account you gave in your Nature Neuroscience article), it reminded me very much of a conversation I had with Oliver Sacks several years ago regarding Virgil.

The basic issue seems to be this: select patients, when thrust into visual input (typically post-surgically) that is not yet integrated with other senses and functions, experience a profound mismatch between habitual information processing and this new flood of input. They must figure out – and if what Kurson related is accurate Mike did this largely on his own with food for thought you fed him – how to filter visual input until it can be reconciled.

The idea I expressed to Oliver, which seems applicable to MM as well, is that we should be able to de-tune visual input until cortical input and networking can better synch with it. The most straightforward (though passive) way to aid this transition is to use a high plus power contact lens (or spectacle lens) to fog input. The progression from "low vision" to "high vision" can be titrated through the amount of lens-induced blur by decreasing the plus power. Further, optometric vision therapy has been used in a variety of clinical presentations to guide perceptual learning, and is compatible with an active approach to titrating this transition.

Sue Barry, Paul Harris and I did a small intro to this on Demo Night. I trust this provides some further food for thought, and it has been a distinct privilege to meet you through VSS. I'd be pleased to discuss the application of these concepts with you at some mutually convenient time.

Sincerely,

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Ione replied that this seemed like a clever idea, but Mike has severe amblyopia. Since his functional visual acuity is so poor she and her colleagues felt that making it any worse, as I had described, would probably not help. Ah, I thought to myself, we're finally getting to the real point**;** what is vision? What does it mean to see, yet alone, see clearly. I decided to pursue the matter further, not from a theoretical standpoint, but in knowing that Ione was going to continue working with Mike. I was hoping that she would feel that clinical optometry might have something to contribute to Mike's rehabilitation. I pressed the issue, and wrote the following:

Perhaps that's the beauty in considering what amblyopia is, where it is, and approaches to resolving the resolution problem as it relates to acuity.

As you noted in the Nature Neuroscience piece, Mike's "optical quality was 20/40 or better." If his deep amblyopia is central in origin, rather than high plus lens fog or some other form of filtration making his acuity worse, it might be a controlled way to allow cortical processing to be more compatible with optical quality.

If my understanding is correct, Mike's prolonged visual deprivation provided the opportunity for re-mapping visual cortex and extrastriate/association-integration areas, particularly since he had become so proficient in his motor and language abilities. Re-developed optical quality can't show up overnight and re-map these areas by eminent domain.

(Forgive my simplistic analogy, but big lottery winners who have a poor sense of finance become confused by their overnight wealth [and worse]. Lottery winners who successfully assimilate sudden wealth typically turn their windfall over to a financial adviser, who guides spending incrementally.)

Are there borderlands between 20/40 optical quality and 20/1000 visual function in which Mike can reside that would allow him to re-map with less conflict? Perhaps giving him less optical quality temporarily might narrow the computational gap, and as central visual cues are re-mapped, optical quality can slowly be improved until the next increment is attained.

Len

Ione politely maintained her ground. She indicated that the presence of high spatial frequencies were essentially invisible to Mike's V1 cells, and thereby to later stages of processing. While it was quite possible that higher level visual areas *could* deal with high spatial frequencies, there was no way of getting that information to these areas because of the bottleneck in V1. I tried my best to elaborate:

Indeed, it's the bottleneck that I'm referring to. If I may, I'd like to go

back to why his basic acuity is so low. Or, why/how high spatial frequencies became invisible to MM's V1. V1 was presumably tuned to high spatial frequencies prior to his accident at age 3. Given that his bilateral corneal opacification rendered him functionally blind, V1 was detuned to high spatial frequencies since that information was no longer coming in.

If my understanding is correct, V1 gets conscripted for other functions rather than remaining dormant. And I would guess that the more time goes by, and the better job MM did in keeping high (frequency) function for somatosensory and auditory/S-L skills, the more challenging it would be for V1 function to be restored to what it was prior to the accident.

If that's correct, then removing the degraded visual input to V1 in one shot creates the processing bottleneck by flooding the pathways with useless higher spatial frequency info. But if instead of flooding V1 with high spatial frequency info, we slowly turned on the tap – going from low spatial frequency, to slightly higher – then we might enable V1 to re-tune to spatial frequency close enough to what's currently visible to allow that to integrate with other neurocognitive information. Then when that small increment is consolidated, go to the next step in higher spatial frequency increments, and so forth.

This way of easing the bottleneck seems consistent with infant visual development/habilitation paradigms, and the general rehabilitative principles used in perceptual re-learning.

Does that sound plausible and/or logical?

Len

I hadn't thought much about our exchange for two years, until an e-mail newsletter from the American Academy of Optometry arrived in my in box on September 11, 2009,[6] announcing this year's plenary session entitled: "Today's Research, Tomorrow's Practice®:Long-term Deprivation and Perception."

The title was virtually the same as Fine's 2003 *Nature Neuroscience* paper about Mike May.[1] Sure enough, Mike and Ione were scheduled to make what I am sure was a memorable presentation to those in attendance. Due to the scheduling conflict of a lecture commitment at OEP's Heart of America Congress, I could not attend. Without having heard his voice, Mike has already spoken inspirationally to me through the articles about him in *Discover* magazine by Michael Abrams.[2,3]

In the more recent *Discover* piece,[3] Abrams elaborates that although Mike has seen faces everywhere since the first day his vision was restored, they simply don't coalesce into recognizable people. Their expressions – their mood and personalities – elude him entirely. He sees no pattern in what the laser interferometer projects clearly onto his retina. These results are backed up by fMRI. The scans show that when May sees faces and objects, the part of his brain that should be used to recognize them is inactive. But, when he sees an object in motion, the motion-detection part of his brain lights up like a disco ball.

Abrams explains that the sudden introduction of a new sense such as sight cannot alter an adult's fundamental way of experiencing the universe.[3] Instead, any new information gleaned from light is grafted onto the original tactile map. Learning how to merge the information between old maps and new is challenging.

Fine has continued to work with May and conduct tests to help measure his brain's ability to interpret space. Although he doesn't get 3-D space, he can do some things that are puzzling. She calls his vision **"**2.5-D." This will be familiar to those who have read the work of David Marr[7] who coined the idea of a 2.5-D sketch of visual space and brings to mind a quote of his that I used in the preface to my book, *Applied Concepts in Vision Therapy*.[8] To paraphrase, studying vision without studying the brain is like trying to understand bird flight by examining only feathers.

Diffusion Tensor Imaging (DTI) studies show how axon fibers link to one another and influence function.[9] DTI studies of May's brain showed that the signals from his eyes do not travel through the usual pathways. Abrams' article reports that one of Fine's collaborators speculates that cells that distinguish objects such as faces were not fully formed in May's brain at age 3, and thus explains his current deficiencies.[3] This sounds unlikely, as one does not have to be a *New York Times* reporter to realize that toddlers are quite skilled in differentiating faces and deciding "who to go to." I suspect that any developmental/behavioral optometrist would agree that while differentiating face perception for certain features may still be developing until age 5 or 6, May's described difficulties far exceeds what one would expect from a 3 year old. It was hard to let this thought go. I went back to Robert Kurson's book and was encouraged by what I re-read. Kurson observes:[4 (p.238)]

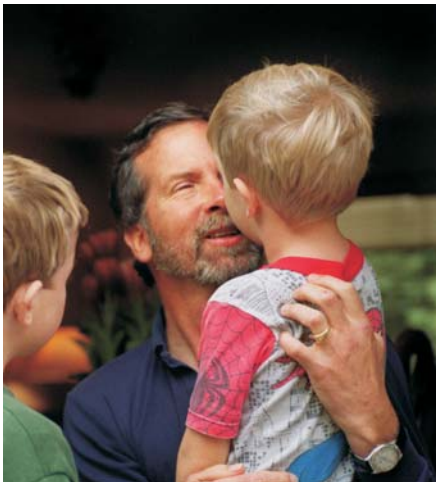
> *How is the very young child to make sense of this jumble of visual data? How is he to translate these shapes into three dimensions and give them meaning, to make them more than just a collection of colorful blobs? How is he to build the knowledge of the world and its objects that is so essential to vision? It's not as if anyone can explain it to him. There is only one way for the very young child to do this. He must interact with the things he sees. He must experiment with them, investigate them, probe them, play with them, touch, taste, smell and hear them. He must handle everything, manipulate everything, go to and reach for everything. He must make his nursery his laboratory.*

In other words, what I think Kurson is saying, is that vision is a learned process, and that movement is an integral part of vision. Sounds vaguely familiar, doesn't it?

Ever hopeful that concepts of developmental and behavioral optometry might be applicable to vision restoration, I glanced again at the 2009 version of Abrams' article in *Discover*.[3] In it he writes:

> *The gift of sight may seem most miraculous, in the end, to those who have never been blind. But May still finds things in the world to entrance him. Sitting in the passenger seat of Fine's car one day, with his dog panting at his feet, he ignores the blue Pacific to the left and the towering, top-heavy eucalyptus trees lining the road like something out of Dr. Seuss. Instead, he gazes at the beam of sunlight filtering through the window onto his lap. 'I can't believe the dust is just floating in the air like this,' he says.*

May's personal discovery of Brownian motion in the Tyndall effect was evocative of the joy Sue Barry found when she dis-



Picture of Mike May seeing his son's blue eyes for the first time.

covered the space between snowflakes.[10] Sue's recounting of her experiences made it clear that stereopsis is not just an epiphenomenon of binocular vision. Mike's visual deprivation, of course, is much more extreme. Sue discovered the palpability of visual space through binocular vision, while Mike struggles to palpate with sight beyond color, movement, and simple shapes or form. Although Mike's case is less about a quale of vision than it is about vision itself, the restoration of any visual function following long-term deprivation can provide insights into the normal development of visual pathways.

Beyond the curiosity and challenge of a singular case, Fine and colleagues have begun to merge their insights on neuroplasticity into the design and functionality of retinal neuroprostheses.[11] With the goal of restoring functional vision in patients with retinal degenerative diseases such as RP, the eyes of blind human subjects are now being implanted with epiretinal prostheses. These prostheses consist of two-dimensional electrode arrays that directly stimulate cells of the neural retina. They enable the retina to intelligently dialogue with the rest of the brain after years of purposeful neglect and speak to the heart of spatiotemporal interactions.

I am sure Mike May and Ione Fine made a big splash at the plenary session of this year's American Academy of Optometry meeting. Perhaps, now, the question posed in the title of essay is more lucid.

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Crashing Through: The Extraordinary True Story of the Man Who Dared to See is available from OEP. It is featured in Product News on page 162 of this *Journal.*