Vision depends on motion: we see things either because they move or because our eyes do. What may be more surprising is that large and miniature eye motions help us examine the world in similar ways—largely at the same time. In this presentation, I will discuss recent research from my lab and others suggesting that exploration and gaze-fixation are not fundamentally different behaviors, but rather two ends of the same visual scanning continuum. They also imply that the same brain systems control our eye movements when we explore and when we fixate—an insight that may ultimately offer clues to understanding not only normal oculomotor function in the healthy brain, but also oculomotor dysfunction in neurological diseases that affect eye movements. This presentation will also include recent data from my lab indicating that primary visual cortical neurons respond differently to object motion versus motion from fixational eye movements.

Commercial Relationships: Susana Martinez-Conde, None

Program Number: 5228
Presentation Time: 3:45 PM–3:50 PM
Introduction to Fixational Eye Movements: Consequences for Vision in Maculopathy, Amblyopia, Traumatic Brain Injury, or Neurodegeneration
Howard S. Ying. Boston University School of Medicine, Boston, MA.

Presentation Description: Fixational eye movements both prevent desensitization of the retina and prevent movement of the target from the fovea. In this mini-symposium, we will explore the consequences of these eye movements in the normal ocular motor system and in various diseases. Amblyopia is an afferent disease where congenital visual obscuration causes maldevelopment of visual processing such that future correction of the obscuration fails to restore vision; however, the interaction between amblyopia and fixation stability is not well-described. Traumatic brain injury most often occurs in a fully-developed and intact ocular motor system, e.g., in young adults, but many of the visual problems that these patients suffer could be attributed to fixation instability. Neurodegenerative diseases such as cerebellar ataxia syndromes at times result in loss of fixation stability through degradation of effenter pathways and offer a window to disease mechanisms. Maculopathies, which result in a deranged fovea, offer a window to compensatory mechanisms.

Commercial Relationships: Howard S. Ying, Lutronics, Inc. (C), SPOUSE – Takeda, Inc., REBIScan, Inc. (C), Johns Hopkins University (P)

Program Number: 5229
Presentation Time: 3:50 PM–4:05 PM
Fixational eye movements
Susana Martinez-Conde.

Presentation Description: Vision depends on motion: we see things either because they move or because our eyes do. What may be more surprising is that large and miniature eye motions help us examine the world in similar ways—largely at the same time. In this presentation, I will discuss recent research from my lab and others suggesting that exploration and gaze-fixation are not all that different processes in the brain. Our eyes scan visual scenes with a same general strategy whether the images are huge or tiny, or even when we try to fix our gaze. These findings indicate that exploration and fixation are not fundamentally different behaviors, but rather two ends of the same visual scanning continuum. They also imply that the same brain systems control our eye movements when we explore and when we fixate—an insight that may ultimately offer clues to understanding not only normal oculomotor function in the healthy brain, but also oculomotor dysfunction in neurological diseases that affect eye movements. This presentation will also include recent data from my lab indicating that primary visual cortical neurons respond differently to object motion versus motion from fixational eye movements.

Commercial Relationships: Susana Martinez-Conde, None

Program Number: 5230
Presentation Time: 4:05 PM–4:20 PM
Disconjugacy of fixational eye movements to detect amblyopia
Kristina Irsch1, 2, 3

1 The Wilmer Eye Institute, The Johns Hopkins University School of Medicine, Baltimore, MD; 2 Institute of the Vision / CIC 1423 / Quinze-Vingts National Eye Hospital, UPMC Sorbonne Universities, Paris, France.

Presentation Description: This talk will go over disconjugate aspects of fixational eye movements as a potential means to detect amblyopia. In particular, we ask whether detection of interocular fixation instability, or variability in alignment of the eyes with respect to one another, could serve as a single sensitive measure for amblyopia, as an alternative to current approaches used in automated screening devices that concentrate on detection of separate amblyopia risk factors. Whether disconjugacy of eye alignment may also be used to guide treatment, notably in preverbal children, as well as assess the efficacy of treatment, will also be discussed.

Commercial Relationships: Kristina Irsch, Patent application (P)

Program Number: 5231
Presentation Time: 4:20 PM–4:35 PM
Characteristics of fixation stability in amblyopia and macular disease
Susana T. Chung.

Presentation Description: Our eyes are constantly in motion even when we attempt to maintain stable fixation on a visual target. These involuntary eye movements during fixation are well characterized for people with normal vision and normal oculomotor control. For people with abnormal fixational eye movements, such as those with strabismic amblyopia or macular disease, the characteristics of their fixational eye movements are less well characterized. In this talk, I will summarize the findings of two studies in which fixational eye movements in human adults with amblyopia or macular disease were recorded using retinal imaging while participants monocularly fixated a fixation cross. Eye position data were recovered using a cross-correlation procedure. Characteristics of slow drifts and microsaccades for the two groups of participants were compared with their respective groups of age-matched controls with normal vision. These comparisons, along with the analyses using a multiple linear regression model to determine the primary factors that limit fixation stability and visual acuity in amblyopic eyes, or in eyes with macular disease, will be presented in the talk.

Commercial Relationships: Susana T. Chung, None

Program Number: 5232
Presentation Time: 4:35 PM–4:50 PM
Fixation instability and amblyopia
Eileen E. Birch.

Presentation Description: Decorrelated visual input from the two eyes during visual development can result in habitual suppression of one eye and amblyopia. Amblyopia can be remediated by rebalancing contrast to overcome suppression, allowing repeated binocular visual experience. Decorrelation during visual development also affects the maturing oculomotor system; amblyopic children have fixation instability. We have identified an association between fixation instability and binocular dysfunction (suppression and abnormal stereacuity). Ongoing studies in our laboratory are quantifying changes in fixation instability in response to monocular and binocular amblyopia treatments. Whether fixation instability poses a limit to visual acuity improvement that could be eliminated with fixation training is unknown. Fixation instability associated with amblyopia may significantly degrade the child’s performance of daily tasks, including fine motor skills and reading.

Commercial Relationships: Eileen E. Birch, None
Fixation Stability: Potential Roles of Microperimetry in the Management of Maculopathy
Quan Dong Nguyen, Truhlsen Eye Institute, University of Nebraska Medical Center, Omaha, NE.

**Presentation Description:** Perimetry has conveyed nondoubtful contributions to the diagnosis and followup of patients. Although well established in the clinical setting, the precise evaluation of macular disorders with conventional perimetry was yet a challenge. The accuracy of the conventional visual field was based on the assumption that the gaze fixation during the examination was stable and located centrally at the fovea. The devices did not detect the eye movements and in those cases with compromised gaze fixation or lack of attention by the patient would have the stimulus presented in a more extensive area than planned for the test. The evaluation of the ability to hold steady fixation, which is a fundamental aspect of good visual function, could not be conducted by means of standard perimetry. Similarly, there were no detection of the preferred retinal locus (PRL) (nonfoveal well-defined region of retina used to fixate a target), no accurate retest examination over the same area, major limitations in patients with low visual acuity, and accurate detection of retinal threshold over discrete retinal lesions smaller than 5° was a known limitation. Current technology of microperimetry has addressed many of the challenges mentioned above. Among the many enhancements, PRL can now be detected. The index presentation will provide overall highlights of microperimetry and its potential roles in the management of macular diseases such as diabetic macular edema, geographic atrophy, and uveitic macular edema.

**Commercial Relationships:** Quan Dong Nguyen, Optos (R), Nidex (R)

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Detecting fixation accuracy/stability using retinal birefringence scanning
David G. Hunter1, 2. 1Ophthalmology, Boston Children’s Hospital, Boston, MA; 2Ophthalmology, Harvard Medical School, Boston, MA.

**Presentation Description:** Foveal fixation can be detected objectively with high precision using retinal birefringence scanning (RBS). Binocular alignment can be assessed with similar speed and accuracy using binocular RBS, which generates a binocularity score of 0-100% after a 2.5 – 5 second bilateral scan. When strabismus is present, the binocularity score is typically 0-20%. Patients with amblyopia unexpectedly have equally low binocularity scores - even when there is no measurable strabismus on cover testing. We will present evidence (from our work and that of others) that the reduced binocularity score in amblyopic patients is the result of fixation instability that develops in the amblyopic eye, and possibly the fellow eye as well, and that stability is restored with successful amblyopia therapy.

**Commercial Relationships:** David G. Hunter, Johns Hopkins University (P), Boston Children's Hospital (P), REBIScan, Inc (I), REBIScan, Inc (C), REBIScan, Inc (S)