Table 1. Childhood Ocular Conditions			
Disease Type	Symptoms	Diagnostic Testing	Treatment
Retinopathy of prematurity (ROP): One of the most common causes of vision loss in childhood. Can lead to lifelong vision impairment. Most commonly found in infants weighing less than 2.75lbs (1250g), or born after fewer than 31 weeks. The smaller a baby is at birth, the more likely that baby is to develop ROP.	Segmented into five stages: Stage I — Mildly abnormal blood vessel growth. Many children who develop stage I improve with no treatment. Stage II — Moderately abnormal blood vessel growth. Many children who develop stage II improve with no treatment. Stage III — Severely abnormal blood vessel growth. The abnormal blood vessels grow toward the center of the eye instead of following their normal growth pattern along the surface of the retina. Stage IV — Partially detached retina. Traction from the scar produced by bleeding, abnormal vessels pulls the retina. Stage V — Completely detached retina and the end stage of the disease.	Fundus examination with binocular indirect ophthalmoscope, lid speculum, scleral depressor and possibly a RetCam (Clarity Medical Systems), as well as optical coherence tomography.	Peripheral laser ablation destroys the peripheral areas of the retina, slowing or reversing the abnormal growth of blood vessels. Patients with ROP are advised to avoid sports with a high risk of head trauma, such as soccer, football and boxing.
Amblyopia	Usually no objective symptoms, making early comprehensive dilated examinations with cyclopentolate vital. Outcomes are best if caught and treated before three years to four years of age. Amblyopia is more challenging to treat after eight years of age.	Cycloplegic refraction, to obtain accurate refraction. Spot Vision Screener (Welch Allyn).	Aggressive and expedient occlusive therapy, with refractive error corrected; including, but not limited to, patching, blur contact lens, patching glasses cyclopentolate/atropine used for occlusion therapy. Vision therapy
Stargardt's disease (Fundus Flavimaculatus) • Mutations in gene ABCA4 are the most common cause of Stargardt's disease. This gene makes a protein that normally clears away vitamin A byproducts inside photoreceptors. Cells that lack the ABCA4 protein accumulate clumps of lipofuscin, a fatty substance that forms yellowish flecks. As the clumps of lipofuscin increase in and around the macula, central vision becomes impaired. Eventually, these fatty deposits lead to the death of photoreceptors, and vision becomes further impaired.	Difficulty with adapting to bright light. Central vision loss. Color vision changes. Stargardt's disease is usually recessive (although there is also a rare dominant inherited pattern). With both parents carrying the mutation, there is a 25% chance of occurrence.	Color vision testing Retinal evaluation	Currently, no cure exists, but promising avenues of research, including gene, stem cell and drug therapies, are in development. Embryonic stem cell treatment is being employed to restore some integrity to the diseased retina in Stargardt's. Anti-VEGF intraocular injections. UV protection, sunglasses and low vision.
Oculocutaneous albinism • Genetic disorder affecting pigmentation of hair, skin and eyes.	 Iris transillumination, nystagmus and amblyopia. Notably fair skin/complexion. White or light-colored hair. 	Careful slit lamp biomicroscope examination with retro illumination for definitive diagnosis. Genetic testing (types I-IV).	Sun protection (patients at risk for melanoma). Recommend Transitions lenses. Prescription sunglasses. Low vision evaluation.