We have all had “that” patient as optometrists; the one who comes in due to sudden onset double vision and whose systemic health history includes any number of serious potential causes. As optometrists it is our duty to either diagnose and manage these patients or to recognize when the cause of their symptoms may be outside our own scope of practice, prompting referral to the correct medical provider. We are often in the unique position to offer meaningful symptom management for these patients once urgent causes have been addressed. Here we will discuss the symptomology, neurological findings, and treatment options available for patients with new onset diplopia through a case example.

Causes of Binocular Diplopia

The causes of binocular double vision are varied, and the likelihood of any particular cause will change depending on the patient’s age. In order to quickly come to the most likely diagnosis it is important to have a better understanding of the most common causes of diplopia, the typical presentations, and the incidence of various origins.
Diplopia (continued)

-Microvascular Causes

Ischemic microvascular disease is one of the most common causes of acute onset diplopia in patients over age 50 years. These patients can present with an isolated cranial nerve 3, 4, or 6 palsy. There are no good prospective population-based studies assessing the incidence of microvascular cranial nerve palsies; however, microvascular causes are estimated to account for approximately 35% of third nerve palsies, 17% of fourth nerve palsies, and 28% of sixth nerve palsies. Although double vision is concerning for patients due to the sudden onset of symptoms, microvascular diplopia nearly always fully resolves within 3 months. Treatment includes minimizing symptoms of diplopia and co-managing with the patient’s primary care physician to address the underlying cause of microvascular ischemia.

Patients with suspected microvascular cranial nerve palsy should undergo the following:

- Laboratory testing, including Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), to rule out giant cell arteritis in patients 55 and older.
- A full ocular health exam checking for any pupil abnormalities, additional ocular motility deficits, and potential muscle restriction or under action.
- In office blood pressure measurement and laboratory testing for fasting blood sugar and HbA1c.

It is often best clinical practice to order neuroimaging in new onset cranial nerve palsy. Neuroimaging is a necessary component of diagnosis in all non-isolated cranial nerve palsies or any cranial nerve 3 palsy unless it completely spares the pupil while completely involving all extraocular muscles innervated by cranial nerve 3. Cranial nerve 6 palsies should be imaged when the patient is younger than 45 years, if the patient is 45 to 55 years of age without vascular risk factors, or when the cranial nerve 6 palsy is associated with severe pain or optic nerve head edema.

-Traumatic Causes

Cranial nerve 4 is the most susceptible cranial nerve to trauma as it has the longest intracranial course and exits the brainstem dorsally at the level of the midbrain; however, cranial nerves 3 and 6 can also be affected by cranial trauma. Trauma accounts for approximately 44% of acquired cranial nerve 4 palsies, with nearly a quarter of these cases having a bilateral cranial nerve 4 presentation. Cranial nerve 6 is especially susceptible to increases in intracranial pressure due to its course over the petrous ridge of the temporal bone. Injuries resulting in intracranial bleeding can result in a cranial nerve 6 palsy due to pressure being placed on the nerve by the hemorrhage.

A full ocular health exam checking for any pupil or additional ocular motility deficits will help rule out causes other than trauma. Blood pressure, fasting blood sugar, and HbA1c may also be necessary if an ischemic cause is suspected. In cases of suspected cranial nerve 4 palsy, a Park’s Three Step should be performed to isolate the involved nerve. A CT may be necessary to rule out fractures related to the trauma or if there is a suspicion of intracranial hemorrhage, which will often be accompanied by additional neurological symptoms.

Figure 1: T2 axial MRI showing white matter lesions in the right cerebral peduncle (arrow)
Giant cell arteritis (GCA) is the most common form of vasculitis in adults over 50 years and constitutes an ocular emergency that needs to be ruled out in the face of acute onset diplopia. The hallmark symptoms include sudden, painless, unilateral vision loss which is often preceded by headache, scalp tenderness, and jaw claudication. If left untreated GCA can cause irreversible unilateral vision loss which can quickly progress to bilateral visual loss.

When assessing a patient for potential GCA, it is critical to assess the patient for the following:

- Possible GCA symptoms.
- Reduced best corrected visual acuity (often count fingers or worse vision).
- Afferent pupillary defect of the affected eye.
- Optic nerve appearance, assessing for pallor, edema, or disc hemorrhages.
- Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Although a high ESR or CRP are not limited to GCA, both being raised gives a 97% specificity for GCA.

Myasthenia gravis is an autoimmune disease in which the body blocks the binding of acetylcholine receptors at neuromuscular junctions causing reduced muscle strength and tone. Although not strictly a disease of the eye or orbits, nearly 75% of patients with generalized myasthenia gravis will have ocular symptoms as their presenting sign, and within two years 85% of those with ocular myasthenia gravis will progress to systemic myasthenia gravis.

These patients will often present with variable double vision that worsens with fatigue or ptosis that is more evident throughout the day. Due to destruction of acetylcholine receptors at the neuromuscular junction, the symptoms will often worsen with prolonged effort. Worsening of the ptosis after prolonged up gaze is a hallmark of this disease.

The following can aid in diagnosing and assessing myasthenia gravis:

- One minute of up-gaze will demonstrate worsening ptosis.
- Application of an ice pack to the affected eye for 2 minutes. A 2 mm reduction in ptosis is indicative of myasthenia gravis.
- Laboratory testing for acetylcholine receptor antibodies.
- Assessment of the patient's swallowing and breathing function to determine possible systemic involvement.
- CT of the chest to rule out thymoma.

Thyroid eye disease is a relatively common autoimmune disorder with a prevalence estimated between 0.5 and 2% and with roughly 50% of patients with Graves disease developing clinically apparent signs and symptoms of thyroid eye disease. Thyroid eye disease is typically associated with hyperthyroidism but often does not occur concurrently with systemic symptoms. In the early stages of the disease patients will complain of foreign body sensation and dry eye symptoms. Upper lid retraction is often the first clinical sign to develop and should be looked for in patients with nonspecific dry eye complaints and a history of thyroid dysfunction.

In later stages of thyroid eye disease, diplopia and exophthalmos can occur secondary to infiltration of the orbital tissue and extraocular muscles by thyroid antibodies. This infiltration and swelling can lead to compression of the optic nerve resulting in optic neuropathy and irreversible vision loss.
Diplopia (continued)

Patients with suspected thyroid eye disease should undergo the following:

- Full ocular health exam evaluating for exposure keratopathy, potential optic nerve compression (resulting in a relative afferent pupillary defect, color abnormalities, and optic nerve pallor).
- Ocular motilities and cover testing to assess potential muscle restriction and ocular alignment.
- Hertel exophthalmometry to assess for proptosis.
- Visual field to rule out scotomas secondary to optic nerve compression.
- Thyroid laboratory testing, including T3, T4, thyroid stimulating hormone, thyroperoxidase antibody, thyroglobulin antibody, and thyroid stimulating immunoglobulin.
- CT of the orbits. Typical CT results will either show thickening of the extraocular muscles without involvement of their associated tendons or increased fat volume in patients with full ocular motility but marked proptosis.

Case Example

AB is an incredibly funny and personable 55-year-old Caucasian male who was referred to our clinic by an outside provider due to a suspected fourth nerve palsy. He had begun noticing vertical binocular diplopia two weeks ago, approximately one week after being in what he described as a minor motor vehicle accident. He had been rear-ended by a car going approximately 20 miles-per-hour while he was stopped in traffic. There was no alteration in level of consciousness, and the paramedics were not called to the scene. He stated that the diplopia was becoming less frequent and smaller in size since the initial onset, but it was especially bothersome in the morning. His wife stated that she had begun noticing a slight droop of his left eyelid with the onset of double vision. Medical history was remarkable for essential hypertension, hypercholesterolemia, type 2 diabetes and keratoconus. He was taking Lipitor, aspirin, fosinopril, metformin, and empagliflozin. His HbA1c had a sudden decrease secondary to a three-week water diet. Ocular history was remarkable for keratoconus for which he was wearing I-Kone contact lenses.

Distance visual acuities with the I-Kone contact lenses were 20/20 right eye and 20/25+2 left eye. Pupils were round but had sluggish constriction in the left eye with resulting anisocoria greater in bright illumination (3.5 mm right eye and 4.5 mm left eye in bright light and 6.5 mm right and left eye in dim illumination). There was no relative afferent pupillary defect. Extraocular muscle movements were full with no restrictions in the right eye. There was -2 underaction of the superior rectus and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye. The patient noted diplopia in superior and inferior rectus of the left eye.

Figure 2: MRA showing narrowing of the left vertebral artery (arrow)
Cover test in various positions of gaze is shown below:

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Due to the vertical strabismus switching from a left hypertropia to a left hypotropia in superior and left gaze, a Park’s 3 Step was not performed.

Anterior segment was remarkable for 1 mm left upper eyelid ptosis. Posterior segment was unremarkable. A reliable Humphrey 30-2 visual field showed scattered defects in a non-neurological in pattern in both eyes.

Discussion

It is easy to forget that many of the tests we do during entrance testing are in fact testing a patient’s neurological function and can lead to a diagnosis of a cranial nerve palsy within minutes of starting the exam. Based on the results of the examination, AB was diagnosed with a partial left cranial nerve 3 palsy. Although he had been referred for a suspected cranial nerve 4 palsy due to the vertical strabismus, the extraocular muscle restriction of the left inferior rectus and left superior rectus, pupil involvement, and ptosis were all consistent with a cranial nerve 3 palsy. In this case determining the diagnosis was straightforward, but determining the cause of the palsy proved more difficult.

AB’s history of diabetes and hypertension and the new onset of the diplopia made a microvascular cause likely; however, he had also been involved in a motor vehicle accident just one week prior to the onset of symptoms. Due to the pupil being affected and the history of trauma, an MRI of the brain and orbits and an MRA of the head was ordered to rule out intracranial bleeding and assess for abnormalities along the pathway of cranial nerve 3. The primary care provider, concerned about the sudden onset double vision, had ordered blood work. The patient’s HbA1c, while relatively controlled at 6.9, had decreased drastically over a short period of time during the water fast. The primary care provider had recently altered the diabetic medications to help stabilize his blood sugar level. The patient also had a normal ESR, CRP, and metabolic panel, ruling out GCA as a potential cause of the diplopia. The patient’s complaint of the double vision being worse in the morning and improving throughout the day made a diagnosis of myasthenia gravis unlikely.

The patient’s MRI showed mild lesions in the midbrain that were felt to be associated with previous ischemic changes (Figure 1). There was no active bleeding or ischemia. His MRA did not show an aneurysm but raised concern over the potential for a left vertebral artery dissection (Figure 2). Due to the poor resolution of the MRA, a CT angiography was ordered which ruled out dissection but did show an abnormal
Diplopia (continued)

appearance of the artery in question (Figure 3). The abnormal appearance of the vertebral artery coupled with the cranial nerve 3 palsy prompted a referral to a practitioner specializing in microvascular disorders in order to better manage AB’s diabetes and hypertension.

AB was followed regularly over the following three months to monitor for any changes in his strabismus and pupil function. During this period AB’s microvascular cranial nerve palsy followed the expected natural progression, with function measurably returning and finally returning to normal approximately 12 weeks after initial onset.

Treatment Options

Monocular Treatment Options

As optometrists we frequently seek to restore the visual system back to its natural state of binocularity; however, in the acute stages of diplopia management, monocular treatment options can be the most comfortable and reliable treatment for the patient. Monocular treatment options allow for easy and cost-effective treatment as the patient progresses through the healing process.

1. Central Occlusion

Central occlusion (Figure 4) blurs the image in one eye, hopefully allowing the patient to ignore the blurred image while attending the remaining single, clear image. This can be helpful in patients with new onset diplopia by reducing symptoms of double vision without negatively impacting peripheral vision. Scotch Tape™ or contact paper provide cosmetically acceptable occlusion at very little cost to either the patient or provider.

2. Sector Occlusion

Sector occlusion is the application of an occluder only in the area of diplopia, allowing the patient to fuse in other directions of gaze. An example of sector occlusion is the occlusion of the lower, nasal portion of the left lens for a left superior oblique palsy.

3. Pirate Patch

Although not the preferred method of patching, as it removes all visual input from the patched eye, the pirate patch is a short-term option to address diplopia for those patients who do not wear glasses. Pirate patches are usually given for the patient to use during specific activities in order to address bothersome diplopia (e.g., watching television). If a pirate patch is used more regularly during treatment it is important to educate the patient about the loss of peripheral vision while patched, and have them alternate which eye is occluded in order to minimize secondary contracture.

4. Monovision Correction

Monovision correction is generally used as a treatment option for long-term diplopia that cannot be managed well with the binocular treatment plans discussed below. Due to the magnification difference induced by different spectacle lenses powers, contact lenses are the
preferred treatment modality for monovision prescriptions. If a patient is comfortable wearing contacts, a monovision contact lens prescription can reduce the perception of the diplopia through suspension of the blurred image and may be helpful in treating diplopia early in the disease process.

For those who have diplopia only at one distance, modified monovision with glasses can be trialed (Figure 5). This type of lens will correct only one eye at the distance with the diplopia, while correcting both eyes for the distances where the patient is able to achieve fusion.

-Binocular Treatment Options

1. Prism

Prism is often not the first line treatment for sudden diplopia caused by a cranial nerve palsy due to the potential for recovery causing changes in the magnitude of deviation. However, prism does allow for binocular vision and improved visual comfort for patients. For those patients who you believe would benefit from prism but are likely to require changes in prism power, Fresnel Press-On Prism™ offers the ability to titrate prism throughout the treatment process. Fresnel Press-On Prism™ is available in a variety of powers allowing you to reduce the prism as the patient heals. It is important to warn patients that the prism will reduce the clarity of their vision, especially at higher prism powers where there is a marked divergence in visual clarity between press-on prism and ground prism.

When prescribing prism in new onset diplopia cases, determining the amount of prism to prescribe can be daunting for some practitioners. Full correcting prism, as determined by the red lens method in a darkened room, can be prescribed for all new onset, constant strabismic patients. Intermittent diplopia is best managed with relieving prism, which can be found by performing the red lens method in normal room illumination.

2. Vision Therapy

Vision therapy can be helpful in regaining function and improving control in new onset diplopia patients. Since there are few sensory anomalies (e.g., suppression or anomalous correspondence) associated with adult onset diplopia, increased motor control and ranges are emphasized during therapy. Therapy activities are begun at distances and positions of gaze that are easy for the patient to achieve fusion and slowly moved towards positions that are more difficult for the patient to maintain motor and sensory fusion.

The Brock string (Figure 6) is a simple vision therapy exercise that provides patients with a sensory cue in order to monitor their motor alignment. As therapy proceeds, patients begin...
moving the Brock string into the position of gaze in which they are most symptomatic and transition from smooth vergence tasks with kinesthetic feedback to vergence jumps controlled by the visual system alone.

3. Surgery

A minimum of six months is needed for the deviation to stabilize before strabismus surgery can be considered. While it is good practice to discuss the possibility of surgery as a potential treatment option, strabismus secondary to a cranial nerve palsy will often partially resolve with time.

Conclusion

Although double vision can be the presenting sign for serious systemic or neurological complications, as optometrists we are uniquely positioned to diagnose, comanage, and treat symptoms. A foundational knowledge of the most common causes of sudden onset diplopia and the relevant testing will allow practitioners to comfortably refer patients who need further workup. However, as reducing the awareness of their double vision is often what is most important to these patients, having an appropriate treatment plan can positively impact patients’ quality of life. Guiding patients in understanding the diagnosis, referring as deemed necessary, and helping manage double vision can be extremely rewarding for both the patient and doctor.

References:

To get your FREE CE certificate for this course:
1. Take the course quiz at [https://online-ce.opt.pacificu.edu/view_course.php?courseid=180](https://online-ce.opt.pacificu.edu/view_course.php?courseid=180) (Log in and Click on “Take Exam”)
2. After taking the exam, add the course to your shopping cart – skip the payment screen
3. Contact JoleneSmith@pacificu.edu to print and send your certificate.

We Welcome Dr. Sarah Lucas

PAULA LUKE, OD, FAAO | VISION THERAPY SERVICE CHIEF

The vision therapy department is proud to introduce the newest member to our team, Dr. Sarah Lucas. As you can see from the above article, Dr. Lucas comes to us with a diverse set of experiences which make her an asset to our team. She graduated from the University of Michigan with a dual major in German Studies and History. She also spent time as part of a team using neuroimaging to research migraines’ impact on pain transmission pathways within the brain. Dr. Lucas always knew she wanted to pursue a career in optometry due to her own personal experience with strabismus and amblyopia. She completed her OD degree at Indiana University School of Optometry in 2018. Dr. Lucas used her desire to help other children with visual disorders and completed the Pediatrics/Vision Therapy residency at Pacific University College of Optometry in 2019. In her free time Sarah enjoys running, hiking, and cross-country skiing with her fiancé. We are excited to have Dr. Lucas continue her time at Pacific University as a faculty member and clinical advisor.
Medical Eye Care Services

LORNE YUDCOVITCH, OD, MS, FAAO | MEDICAL EYE CARE SERVICE CHIEF

The College of Optometry EyeClinics have a variety of specialized diagnostic testing available for patients in the Medical Eye Care/Ocular Disease Service.

Special testing includes:

- Ocular Biometry
- OCT and OCT Angiography
- Specialized Color Vision Tests
- Specialized Visual Field Testing
- Anterior Segment Photography
- Glare and Potential Acuity Testing
- A-scan and B-Scan Ultrasonography
- Preferential Hyperacuity Perimetry
- Fundus Autofluorescence Photography
- Corneal Topography/Thickness Mapping
- Macular Pigment Optical Density Testing
- Eyelid Lesion Evaluation/Biopsy
- Ocular Electrodiagnostic Tests (VEP, ERG, EOG)
- Meibomography and Dry Eye Biomarker Tests

Eyelid lesion evaluation and biopsy, as well as electrodiagnostic testing, are performed at our Hillsboro EyeClinic by Dr. Blair Lonsberry and Dr. Denise Goodwin, respectively. Specialized dry eye testing is performed in our Beaverton EyeClinic Dry Eye Solutions service by Dr. Tracy Doll.

Please feel free to contact us at any of our EyeClinics should you need to refer a patient for specialized ocular disease testing.

Multifocal Lenses & Ocular Dominance

MATT LAMPA, OD, FAAO | CORNEA AND CONTACT LENS SERVICE CHIEF

As the add power necessary to provide optimal near vision with multifocal contact lenses increases distance visual function may be impeded. One of the common troubleshooting techniques found in multifocal contact lens manufacturers fitting guides is to break from traditional spectacle lens prescribing habits and utilize dissimilar add powers in each eye. To determine over which eye to put the higher add, a technique known as the swinging plus test may be utilized. The test is performed with the optimal distance correction (either single vision contact lenses or spectacle lenses) in place. A loose trial lens of +1.50 D is held over one eye at a time while both eyes are open and sighting a distance chart with a grouping of letters near their best corrected visual acuity. The higher multifocal add power should be placed over the eye that noted less disruption to their distance vision. This method is likely to provide the patient with the near vision they desire without overly sacrificing their distance vision.
Intense Pulsed Light Therapy

TRACY DOLL, OD, FAAO | PACIFIC DRY EYE SOLUTIONS COORDINATOR

Pacific Dry Eye Solutions is thrilled to offer new treatment for ocular surface dryness: intense pulsed light therapy with the M22 (by Lumenis). Intense pulsed light therapy is indicated to treat ocular rosacea, meibomian gland dysfunction, chronic inflammatory dry eye, and Demodex blepharitis.

In rosacea, meibomian gland dysfunction, and chronic inflammatory dry eye, small telangiectatic blood vessels infiltrate the lid margin, increasing inflammatory blood cell delivery. Intense pulsed light therapy causes coagulation and regression of these small blood vessels, resulting in reduction of inflammatory blood cell delivery to the ocular tissues.

As an added effect, the chitinous exoskeletons of Demodex folliculorum and Demodex brevis mites are not built to withstand the intensity of this light therapy. The immediate population reduction of Demodex mites is an effective treatment for blepharitis. Patients can experience reduced symptoms of dryness and redness of the eyelids, as well as improved quality of meibomian gland secretion and decreased inflammatory mediators on the ocular surface.

Treatment of ocular surface conditions with intense pulsed light therapy is comprised of a series of 3 to 5 sessions spaced 3 to 4 weeks apart. Most patients begin to notice improvement in signs and symptoms after 2 to 3 treatments, but some patient feeling relief after the very first in-office treatment. It is recommended that the intense pulsed light therapy series be repeated every 6 to 12 months to maintain results.

A total of about 30 triple pulses, at 590 nm is applied in a W pattern on the skin under the eye from ear to ear and back again. With protective eye shields in place, an additional eyelid treatment can be applied at the end of the treatment protocol for patients with severe presentations. It should be noted that intense pulsed light therapy for meibomian gland dysfunction is usually performed in conjunction for with meibomian gland expression for maximal results.

There is minimal recovery time, with the patient being able to return to work and daily activity immediately.

If you would like to refer your patient for intense pulsed light therapy, call Pacific Dry Eye Solutions at 503-352-1699.
Dr. Lowery Inducted to Hall of Fame

CHRISTI CLOSSON, OD, FAAO | LOW VISION SERVICE CHIEF

In May, our own Dr. John P. Lowery was inducted into the Oregon Lions Foundation for Sight and Hearing Hall of Fame! Dr. Lowery has been on faculty at Pacific University College of Optometry for 22 years and has served as the Chief of Pediatrics for 20 years.

In 1995, Dr. Lowery became the Clinic Director of the Statewide Low Vision Clinic through a partnership with the Oregon Lions Sight & Hearing Foundation. Every year, he conducts up to 30 FREE clinic events for visually impaired children from birth to 21 years of age. 70% of these children have multiple disabilities, and the majority are from low income families who do not have access to educationally-focused low vision evaluations. Dr. Lowery’s pediatric and medical expertise combined with his educational perspective creates a collaborative setting that emphasizes the needs of the students with low vision.

Each year, Dr. Lowery travels approximately 4000 miles to provide clinic services in all parts of the state and has now provided over 3500 evaluations!

When asked about Dr. Lowery, Dean Jennifer Coyle said, “I have been lucky enough to know Dr. Lowery for 30 years as my classmate in optometry school. He has always been dedicated to serving children and other underserved populations. He has a very special gift that has been recognized and celebrated through this award, and I couldn't be more proud to be his fellow alumna at Pacific University!”

Congratulations Dr. Lowery for an honor well deserved and sincere thanks for taking care of the needs of these students with low vision. You are an inspiration to us all!

Neuro-ophthalmology Resources

DENISE GOODWIN, OD, FAAO | NEURO-OPHTHALMIC DISEASE CLINIC COORDINATOR

Want to learn more about neuro-ophthalmology topics? NOVEL (Neuro-Ophthalmology Virtual Education Library) is a free open access repository of images, video, PowerPoints, and articles from some of the leaders in neuro-ophthalmology. It can be found at https://novel.utah.edu/.

Another great resource is http://www.neuroophthalmology.ca/. This covers very interesting vision loss, eye movement, and pupil cases from the novice to expert level and allows you to quiz yourself to assess understanding.

Looking for information about neuroimaging? A great resource is at https://medpix.nlm.nih.gov. Here there are hundreds of cases. Try identifying the type of scan and abnormality. Click on the blurry words under the scan to test your knowledge. The more you do, the better you become at reading the scans.
Practice Management Tips

CINDI RAPP, RDH | DIRECTOR OF CLINICAL OPERATIONS

Supervisor? Coach? Leader? Practice managers wear many hats!

A supervisor gives answers. A coach gives the work of finding the answers back to the employee. A leader provides inspiration and direction. Our “hats” change many times throughout the day based on all the various activities and issues in the workplace. Many managers instinctively think their job is to solve issues brought to them by employees. But often, when this is done, the next challenge that arises will have the employee back in the manager’s office looking for yet another answer.

Instead, we have the opportunity to help strengthen the mindset of critical thinking skills by gently sending the issue back to the person. We do this, not because we can’t or don’t want to solve the issue, but because in doing so, we are preventing them the opportunity to discover their own ability to solve the issue. If you put on your “coaching” hat and begin asking leading questions, discovering what the employee really wants, and gently guiding them to their own answers and resourcefulness, their increased confidence encourages them to find solutions on their own.

It takes repetition, but continue to give the work back to the employee. Assist them in thinking through the people, organizations, publications, policies, courses, etc. that might be resources for solutions or answers to their questions. Help them to learn how to get creative about solving challenges and strengthening their skillset for independence and self-reliance. Refrain from doing all the work for them. See your role as both that of a coach and a leader. Coach people to independence and feelings of pride. Be the leader and demonstrate what matters most in relationships at work: the experience of feeling valued and connected and making others around you better!

Reminder: Post your July 1, 2019 - June 30, 2020 BOLI Poster, you can get it here www.oregon.gov/boli/TA/Pages/Req_Post.aspx

CE Opportunities

September 2019:
- PCLI CE: Cataracts - Eight Cutting Edge Topics; Portland, OR; September 19.

October 2019:
- GWCO; Oregon Convention Center, Portland, OR; October 10-13.
- American Academy of Optometry; Orlando, FL; October 23-27.

November 2019:
- PCLI CE: MIGS - Changing Paradigm for Glaucoma Management and Glaucoma Treatment Considerations; Portland, OR; November 12.

January 2020:

March 2020:
- Teplick Vision’s 26th Annual CE Event; Portland, OR; March 8, 2020; for more information, email Teri.cummings@nvisioncenters.com.

April 2020:
- Coeur d'Alene CE Event; Coeur d'Alene Resort, Coeur d'Alene, ID; April 24-25, 2020.
Referral Service Contact Numbers

**Pacific EyeClinic Forest Grove**
2043 College Way, Forest Grove, OR 97116
Phone: 503-352-2020; Fax: 503-352-2261
- Vision Therapy and Pediatrics: Scott Cooper, OD; Graham Erickson, OD; ChunMing Liu, OD; JP Lowery, OD; Sarah Lucas, OD; Paula Luke, OD
- Medical Eye Care: Ryan Bulson, OD; Lorne Yudcovitch, OD
- Low Vision: Karl Citek, OD; JP Lowery, OD
- Contact Lens: Mark Andre; Tad Buckingham, OD; Patrick Caroline; Emily Cheng, OD; Bill Hefner, OD; Beth Kinoshita, OD; Matt Lampa, OD

**Pacific EyeClinic Cornelius**
1151 N. Adair, Suite 104 Cornelius, OR 97113
Phone: 503-352-8543; Fax: 503-352-8535
- Pediatrics: JP Lowery, OD
- Medical Eye Care: Tad Buckingham, OD; Bill Hefner, OD; Caroline Ooley, OD; Lorne Yudcovitch, OD

**Pacific EyeClinic Hillsboro**
222 SE 8th Avenue, Hillsboro, OR 97123
Phone: 503-352-7300; Fax: 503-352-7220
- Pediatrics: Ryan Bulson, OD
- Medical Eye Care: Dina Erickson, OD; Michela Kenning, OD; Hannah Shinoda, OD
- Periocular and Eyelid Services: Blair Lonsberry, OD; Lorne Yudcovitch, OD
- Neuro-ophthalmic Disease: Denise Goodwin, OD

**Pacific EyeClinic Beaverton**
12600 SW Crescent St, Suite 130, Beaverton, OR 97005
Phone: 503-352-1699; Fax: 503-352-1690
- 3D Vision: James Kundart, OD
- Pediatrics: Alan Love, OD
- Medical Eye Care: Susan Littlefield, OD
- Contact Lens: Matt Lampa, OD
- Dry Eye Solutions: Tracy Doll, OD

**Pacific EyeClinic Portland**
511 SW 10th Ave., Suite 500, Portland, OR 97205
Phone: 503-352-2500; Fax: 503-352-2523
- Vision Therapy and Pediatrics: Ben Conway, OD; Paula Luke, OD; Elizabeth Powers, OD
- Medical Eye Care: Scott Klemens, OD; Scott Overton, OD; Carole Timpone, OD
- Contact Lens: Mark Andre; Emily Cheng, OD; Matt Lampa, OD; Scott Overton, OD; Sarah Pajot, OD; Neeru Shore, OD
- Neuro-ophthalmic Disease/Strabismus: Paula Luke, OD
- Low Vision: Scott Overton, OD

**Pacific EyeClinic Vancouver**
2214 E. 13th Street, Suite 212, Vancouver, WA 98661
Phone: 360-947-3302; Fax: 360-737-2120
- Low Vision Service only: Christi Closson, OD