CJO RCO

CANADIAN JOURNAL *of* OPTOMETRY | REVUE CANADIENNE D'OPTOMÉTRIE

EST. 1939 VOLUME 79 NUMBER 3

CASE STUDY

Optometric Management of a Third Nerve Palsy in a Prison Setting

CLINICAL RESEARCH

Idiopathic Juxtafoveal Telangiectasia

INNOVATIONS

Innovation Eye Care: Sports Vision

PRACTICE MANAGEMENT

Every Member of Your Team is a Salesperson

CANADIAN ASSOCIATION OF OPTOMETRISTS
ASSOCIATION CANADIENNE DES OPTOMÉTRISTES

DISCOVER THE POWER AND SIMPLICITY OF SYSTANE®

ONE Name, **ONE** Brand, **ONE** Recommendation

Systane®

The brand Eye Care Professionals have made Number ONE[1]

The one brand with a comprehensive portfolio of products designed to meet the unique needs of your different patients, your practice and your commitment to care.

Visit: www.systane.ca to learn more.

NOVARTIS

Reference: 1. Drugstore Canada and L'actualité pharmaceutique 2016 Survey on OTC Counselling and Recommendations and The Medical Post and L'actualité médicale 2016 Survey on OTC Counselling and Recommendations. AC Nielson Track latest 52 weeks ending April 2, 2016.

Alcon A Novartis Division

CANADIAN JOURNAL *of* OPTOMETRY
REVUE CANADIENNE D'OPTOMÉTRIE
Vol 79, No 3
Fall/Automne 2017
ISSN 0045-5075
The *Canadian Journal of Optometry* is the official publication of the Canadian Association of Optometrists (CAO) / La Revue canadienne d'optométrie est la publication officielle de l'Association canadienne des optométristes (ACO) : 234 Argyle Avenue, Ottawa ON, K2P 1B9. Phone 613 235-7924 / 888 263-4676, fax 613 235-2025, e-mail info@opto.ca, website www.opto.ca. Publications Mail Registration No. 558206 / Envoi de publication – Enregistrement no 558206.
The *Canadian Journal of Optometry / La Revue canadienne d'optométrie* (USPS#0009-364) is published four times per year. Address changes should be sent to CAO, 234 Argyle Avenue, Ottawa, ON K2P 1B9.

Published by:

maracle
create • print • integrate

maracleinc.com

CJO RCO
CANADIAN JOURNAL *of* OPTOMETRY | REVUE CANADIENNE D'OPTOMÉTRIE
EST. 1939 VOLUME 79 NUMBER 3

CONTENTS

On the Cover

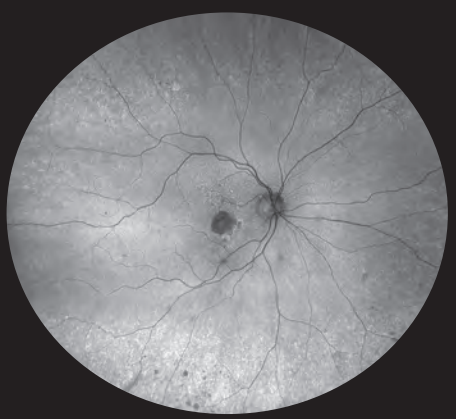
In Canada, the CSC contracts optometrists as part of their duty of care to offenders, to maintain ocular health.

▶ optomap® imaging takes less than half a second...

TECHNOLOGICAL INNOVATION
to help prevent vision loss

optomap non-mydriatic ultra-widefield technology delivers detailed 200° high resolution images in less than half a second. **This technology can image pathology past the vortex vessels, helping you find disease sooner and manage it more effectively**

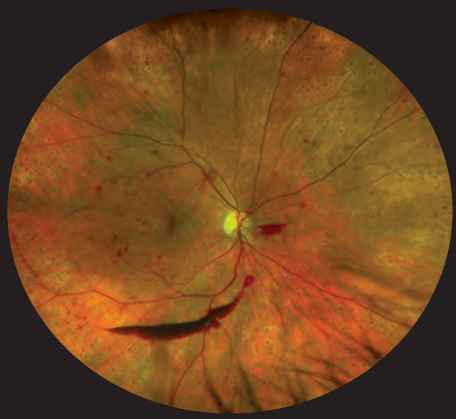
the ONLY 200° single-capture *af* image



PRACTICE EFFICIENCY
to improve practice flow

optomap imaging is so fast and easy it can speed practice flow giving you more time for high value activities. **Routine use of optomap can improve and increase patient throughput and potentially create an additional revenue stream**

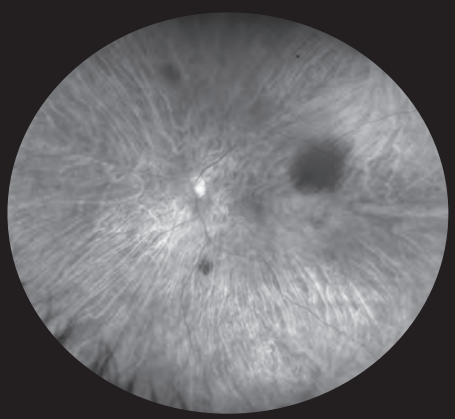
the ONLY 200° single-capture *color* image



CLINICAL OUTCOMES
to uncover critical information

optomap ultra-widefield imaging is a proven tool for effective clinical decision making. **More than 400 peer reviewed studies show the value of optomap imaging in diagnosis, treatment planning, and patient engagement**

the ONLY 200° single-capture *choroidal layer* image



Contact us for your risk-free evaluation at 800-854-3039 or BDS@optos.com

Nikon A Nikon Company

optos®
Building *The* Retina Company

optos.com

Image Courtesy Paulo Stanga, MD

B. Ralph Chou, MSc, OD, FAAO
Editor-in-Chief

The summer of 2017 has been eventful for several reasons. It began with the CAO Congress in Ottawa during the last week of June. The many high-quality educational sessions were punctuated with social events where I talked with many former students. I also enjoyed seeing many members of the UW Optometry class of 1979. It's hard to believe that my very first CAO Congress was 38 years ago in Edmonton.

Next was the official arrival of Dr. Stanley Woo as Director of the School of Optometry and Vision Science at the University of Waterloo on 1 July. He will preside over the School's 50th Anniversary celebrations this September. Running a complex operation like the School of Optometry is a challenge in the best of times, but Dr. Woo will also be contending with renewal of the School's accreditation with the AOA's Accreditation Council on Optometric Education and updating of the OD program's curriculum in light of recent and possible future changes in our profession's scope of practice. I wish him well.

As I write this editorial, I am taking a break from the many requests over the last couple of weeks for interviews from across Canada and the USA about eye safety for the solar eclipse of 21 August. By the time you read this editorial, the eclipse will be history, and its impact on the population of North America is hard to predict. If it is a clear day, virtually all of the population of both Canada and the USA will see at least a deep partial eclipse, while a narrow strip of the continental USA will experience the first total eclipse over North America since 1979. For once, astronomers, optometrists and ophthalmologists united to provide the same advice to the public about safe viewing practices. This is something I have been working on for many years, and I hope many of you were able to take a few moments to experience the eclipse that day.

Our lead article by Dr. Tousignant addresses the need for vision care of inmates in correctional facilities. Providing eye care in a prison setting definitely has its challenges. I welcome Dr. Graham Erickson of the Pacific University College of Optometry, whose first article on advances in Sports Vision appears in this issue. Finally I would like to thank everyone involved in assembling our first Supplement for 2017. The evidence-based guideline on management of glaucoma is an important contribution and represents months of work. I hope you will enjoy reading it. ●

The leading cause of ocular discomfort and contact lens dropout is dryness.

The TearScience® solution for MGD allows eye care professionals to safely and effectively identify, diagnose and manage MGD.

.

"Evaluation of meibomian glands is now standard in my patient workups"

Steven Ferguson, OD
Dunes Eye consultants

TearScience®

CLARION®
MEDICAL TECHNOLOGIES

YOUR DRY EYE SOLUTION PROVIDER

Clarion Medical Technologies is proud to offer the most comprehensive line of products for the management and treatment of dry eye and MGD.

Call us today to find out how to provide relief for your patients.

800-668-5236 | www.clarionmedical.com | info@clarionmedical.com

B. Ralph Chou, MSc, OD, FAAO
Rédacteur en chef

L'été 2017 a été riche en événements. Tout d'abord, il y a eu le congrès de l'ACO, qui s'est déroulé à Ottawa durant la dernière semaine de juin. En plus du grand nombre de séances instructives de qualité supérieure, des événements sociaux ont été tenus, durant lesquels j'ai pu m'entretenir avec plusieurs anciens étudiants. J'ai également eu le plaisir de revoir de nombreux membres de la promotion de 1979 du programme d'optométrie de l'Université de Waterloo. Il est difficile de croire que j'ai assisté à mon tout premier congrès de l'ACO il y a 38 ans, à Edmonton.

Ensuite, il y a eu l'arrivée officielle du Dr Stanley Woo à titre de directeur de l'École d'optométrie et des sciences de la vision à l'Université de Waterloo le 1er juillet. Au cours du mois de septembre, le Dr Woo présidera les célébrations du 50e anniversaire de l'École. La gestion d'une organisation complexe comme l'École d'optométrie représente déjà un défi dans les circonstances les plus favorables. Or, le Dr Woo devra aussi s'occuper du renouvellement de l'attestation professionnelle de l'École auprès de l'Accreditation Council on Optometric Education (ACOE) de l'American Optometric Association (AOA) et de la mise à jour du contenu du programme de doctorat en optométrie à la lumière des changements récents et éventuels dans le champ d'exercice de notre profession. Je lui souhaite bon succès.

En rédigeant mon éditorial, j'ai mis temporairement de côté les nombreuses demandes d'entrevue que j'ai reçues au cours des dernières semaines provenant de partout au Canada et aux États-Unis concernant la sécurité oculaire dans le contexte de l'éclipse solaire du 21 août. Lorsque vous lirez ces lignes, l'éclipse sera chose du passé, et il est difficile de prédire ses effets sur la population de l'Amérique du Nord. Si le temps est clair, presque toute la population du Canada et des États-Unis pourra observer au moins une éclipse solaire partielle, tandis qu'une éclipse totale, la première au-dessus de l'Amérique du Nord depuis 1979, sera visible d'une bande étroite de la partie continentale des É.-U. Pour une fois, les astronomes, les optométristes et les ophtalmologistes se sont entendus pour donner les mêmes conseils au public au sujet des pratiques sécuritaires d'observation. C'est un dossier sur lequel je travaille depuis de nombreuses années, et j'espère que bon nombre d'entre vous ont eu la chance de prendre quelques instants pour contempler l'éclipse.

Notre article principal, écrit par le Dr Tousignant, traite de la nécessité d'offrir des soins de la vue aux détenus dans les établissements correctionnels. La prestation de soins oculaires dans une prison porte certainement son lot de défis. Je souhaite la bienvenue au Dr Graham Erickson, de la Pacific University College of Optometry, dont le premier article sur les progrès relatifs à la vision dans les sports est publié dans le présent numéro. Enfin, j'aimerais remercier tous ceux qui ont participé à la production de notre premier supplément de 2017. Le guide sur la gestion du glaucome, qui est fondé sur des faits, est une importante contribution et représente des mois de travail. J'espère que vous vous plairez à le consulter. ●

ACUVUE®
BRAND CONTACT LENSES

MARKETING MADE EASY

INTRODUCING
YOUR ONE-STOP SHOP
TO ENHANCE YOUR PRACTICE'S
DIGITAL PRESENCE

ACUVUELIBRARY.CA

Optometric Management of a Third Nerve Palsy in a Prison Setting

Benoît Tousignant, OD MSc MPH FAAO
Assistant Professor
École d'optométrie,
Université de Montréal

Abstract

Third nerve palsy (TNP) is a neuro-ophthalmic condition that is likely to be encountered by optometrists in various clinical settings. It affects extraocular muscles and may impair eyelid and pupil functions to various degrees. Its aetiology in adults is often linked to compressive lesions of the central nervous system or to vascular ischemic causes from systemic disease such as diabetes and hypertension. This case report presents the management of a TNP in a multidisciplinary Canadian prison setting, where the patient population is often underserved. Current practices for neuroimaging cases of TNP in a conventional clinical setting are presented. The challenges and advantages of management of TNP in a prison setting are discussed, highlighting the particularities of caring for the vulnerable patient population in the prison setting.

KEY WORDS:
Third nerve palsy, prison, interdisciplinary care, neuroimaging, underserved population

INTRODUCTION

Third cranial nerve palsy affects extraocular muscles, the eyelid and the pupil, and is encountered by many optometrists. This case highlights the particularities of its management in a multidisciplinary prison setting.

CASE REPORT

A 60-year-old Caucasian male presented to the optometry service of a Canadian medium-security penitentiary after being referred by the prison's mental health facility physician for "reduced and/or double vision".

Upon questioning, the patient mainly complained of blurry vision since having lost his spectacles. He denied the presence of diplopia, pain, headaches and ocular trauma. The patient's responses were somewhat limited by his mental health condition and a speech impediment. He had undergone cataract surgery (2009) and usually wore spectacles to correct compound hyperopic astigmatism (2011). Binocular vision and ocular health were otherwise unremarkable.

His current medical history included type-2 diabetes (1999, recent HbA1c 10.1%), obesity, hypertension, hypercholesterolemia, benign prostatic hyperplasia, diabetic neuropathy, sleep apnoea, stuttering, anaemia, slight intellectual disability and paranoid schizophrenia. He was being treated with gabapentin, olanzapine, valproate, atorvastatin, ezetimibe, furosemide, irbesartan, nifedipine, insulin, ferrous sulfate and terazosin. Neither the family medical history nor the family ocular history were available.

External exam revealed complete ptosis OD, which had not been mentioned by either the patient or a physician. Uncorrected distance visual acuities were OD 6/21 (holding eyelid) and OS 6/12, with inconclusive pinhole testing. Manifest distance refraction showed a stable compound hyperopic prescription, which corrected visual acuities to

OD 6/6 (holding eyelids and OS 6/7.5. Cover test was inconclusive (comprehension/fixation difficult), but ocular alignment by Krimsky reflexes showed a 30 prism dioptre exotropia and 10 prism dioptre hypotropia OD. In the right eye, adduction, depression and elevation showed grade -4 limitations, with full motility OS in all directions. Pupils were equal and reactive to light and accommodation, without afferent pupillary defect. Anterior segment examination revealed complete ptosis OD, but was otherwise unremarkable. Intraocular pressure was 14 mmHg OU. The left intraocular lens had slight posterior capsule opacification. Dilated fundus examination was unremarkable, with no evidence of diabetic retinopathy. Automated visual field testing was not available, and confrontation visual fields were unreliable.

This clinical portrait suggested a complete, isolated, pupil-sparing TNP, supported by the following: unilateral, painless, atraumatic, recent-onset exodeviation and hypodeviation, complete ptosis, age above 50, normal pupillary function and normal function of the fourth and sixth cranial nerves. Diabetes mellitus was considered to be the most likely cause, given the patient's glycaemic profile and well-controlled systemic hypertension. The prison physician was consulted, which prompted a referral for neuroimaging to rule out compressive aetiologies. For the following week, the nursing staff was advised to monitor the patient daily for late-onset right pupil dilation. A follow-up optometry appointment was scheduled three weeks later. New spectacles were ordered, but no diplopia management was deemed necessary from the complete ptosis. The patient was counselled on the importance of glycaemic control and the effects of diabetic eye disease. The prison nutritionist was solicited to reinforce dietary recommendations.

At the follow-up exam, the patient was more alert, which facilitated history-taking. Upon questioning, he denied diplopia and pain, and seemed content with the new correction. Neuroimaging results were not yet available. The patient record showed no changes in pupil function. Corrected visual acuities were OD 6/6 and OS 6/7.5. External exam showed no ptosis. The patient's eyes were now aligned in primary gaze, with a 30 prism dioptre exophoria at distance and near, normal sensory fusion (red filter) and stereopsis. Extraocular motilities were full in all directions. Pupillary reflexes were normal, without anisocoria. A follow-up examination was planned for two months later. The patient was advised to alert the nursing staff of any diplopia, or any changes in vision or pupil/eyelid appearance.

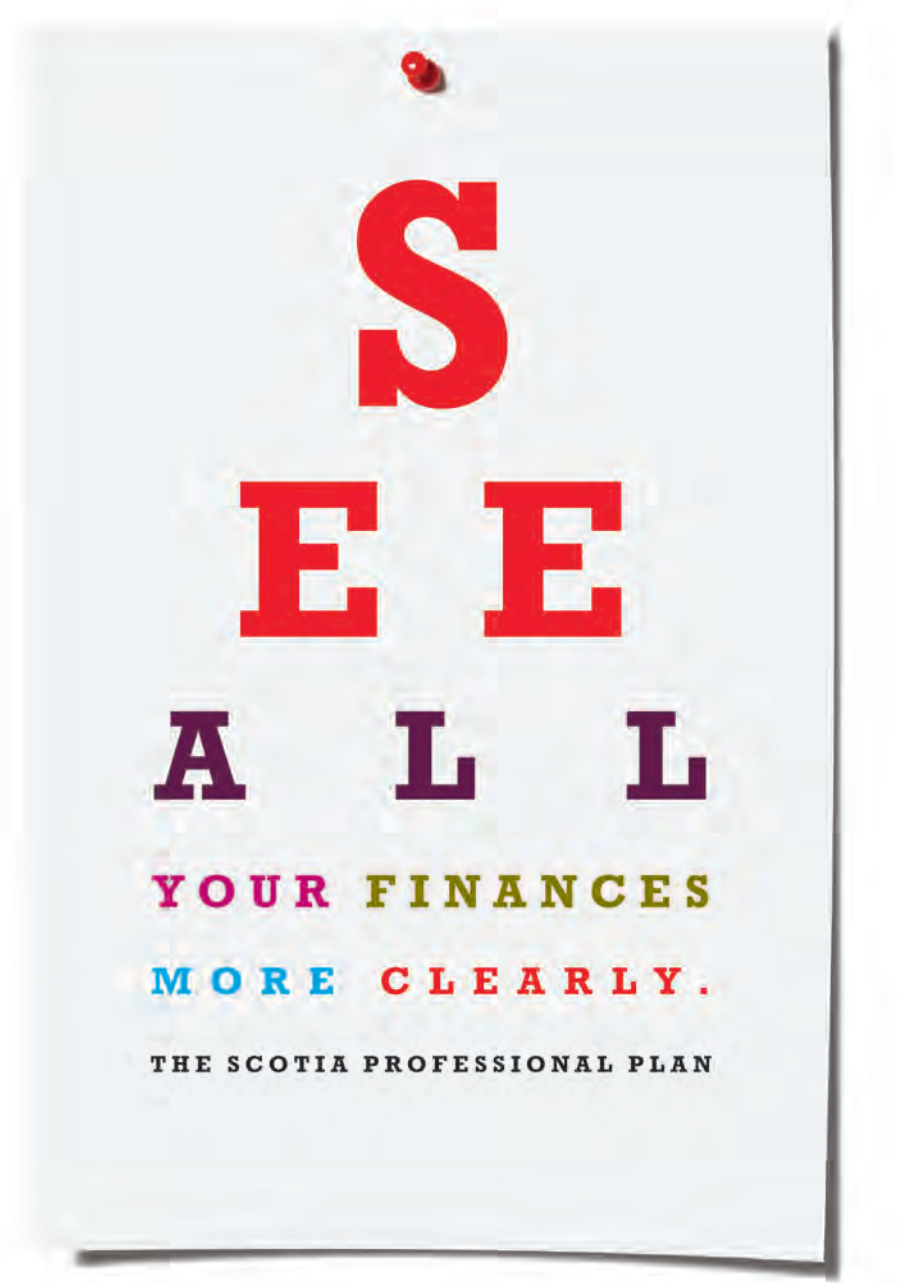
At the two-month follow-up exam, the patient reported no change in his condition. Corrected visual acuities were stable and an external exam was normal. The patient was now orthophoric at distance and near. Extraocular motilities were full in all directions and pupil reflexes were normal. The radiology report included a computed tomography (CT) scan of the head and orbits, with contrast, showing no intracranial lesions or significant anomalies. The patient was educated on his apparent complete recovery and advised to return in two years, in accordance with the Correctional Service of Canada (CSC) policy on the frequency of eye examinations.

DISCUSSION

Clinical presentation and pathophysiology

The third cranial nerve innervates the superior, inferior and medial recti muscles, as well as the inferior oblique and levator palpebrae. Its external and dorsolateral fibres control accommodation and miosis. As such, TNP usually presents with ipsilateral ptosis, hypodeviation with concurrent exodeviation, diplopia, a possible fixed and mydriatic pupil, and blurry vision.[1,2] Complete TNP includes the inability to elevate, depress or adduct the affected eye, along with a complete ptosis. Pupil involvement depends on the aetiology. Partial or incomplete TNP will restrict extraocular motilities to various degrees, impairing one or more extraocular muscles, and cause variable pupil involvement. Partial palsies most often affect either the inferior branch of the nerve (medial rectus, inferior rectus and inferior oblique) or its superior branch (superior rectus and levator).[1,3] Most cases of TNP are unilateral – bilateral cases occur only with central oculomotor nucleus involvement.

Principal aetiologies for TNP include vascular or ischemic causes (diabetes, hypertension, atherosclerotic disease), compression (aneurysm, tumour), trauma and congenital.[1,3,4] Factors that determine the aetiology include pupil involvement, pain, vascular risk factors and age. Mydriasis will typically indicate a compressive lesion.[1,3] As this nerve travels from the midbrain to the orbit through the subarachnoid space, it passes next to the Circle of Willis, close to the posterior communicating artery, where an aneurysm may compress the nerve's external pupillary fibres, resulting in mydriasis and loss of pupil function. Compressive palsies often present with pain or headache.[3,4] Ischemic causes, conversely, will typically only affect internal third nerve fibres, resulting in a painless, pupil-sparing presentation.[4,5] In patients above age 50, TNP is more likely ischemic in nature, where the *vasa nervorum* may suffer ischemia, affecting the main internal fibres that it supplies. Cases under age 50 are more likely to have compressive aetiologies.[1] TNP may be combined with fourth, fifth and/or sixth nerves palsies with lesions affecting the cavernous sinus.[6]



The Scotia Professional Plan for Optometrists.

You've worked hard to get where you are today and we can help ensure your ongoing success. The *Scotia Professional*® Plan lets you manage your professional and personal banking with a customized suite of products and services, preferred rates, and the support of a dedicated Scotiabank advisor. It's the best way to bring all your finances into focus.

To learn more, visit your nearest branch or www.scotiabank.com/professional

You're richer than you think.® Scotiabank®

Differential diagnosis

In this case, differential diagnoses included myasthenia gravis, partial TNP and a pre-existing or decompensated deviation with concurrent ptosis. The latter was excluded based on previous records that showed normal ocular alignment and eyelid function. A partial palsy was excluded based on complete impairment of the eyelid and extraocular muscles. Lastly, ocular myasthenia gravis may present with diplopia and involve extraocular and/or levator muscles. However, muscular imbalances will typically increase with muscle fatigue. Here, it was excluded, since ptosis was full and constant, with both adduction and elevation completely impaired.

Case management

The recovery period for TNP from suspected vascular causes is typically between three and 12 months.[1,3] Its management involves controlling diplopia symptoms, monitoring for pupil involvement, neuroimaging and targeting the underlying systemic condition.[3,4] Diplopia management involves patching or using a frosted lens or Fresnel prisms to minimize symptoms.[3,4] While Fresnel prisms are inexpensive and simple, ordering a frosted lens requires more expense and delays for a likely transient solution. In this case, although history-taking was unreliable, complete ptosis eliminated potential complaints of diplopia, and when ptosis had receded, the patient had also recovered sensory fusion.

A pupil-sparing, presumed ischemic TNP should be closely monitored for pupil involvement during the first week, as some compressive causes initially present with normal pupil function.[1,3,6] Here, this was facilitated by the fact that the nursing staff was present on a daily basis.

Role of neuroimaging

The evidence based on neuroimaging for presumed ischemic TNP in patients over age 50 has evolved over time and is still debated.[7,8] The main issue regards the diagnostic value of neuroimaging to rule out aneurysm or other compressive lesions in such cases. Current practices in ophthalmology have evolved from careful observation (3 to 6 months) to performing magnetic resonance imaging (MRI) and angiography (MRA) for most patients with TNP, to rule out compressive lesions more effectively.[6,9–11] Despite a current lack of high-level evidence (systematic review, meta-analyses or evidence-based clinical guidelines) regarding this matter, prospective studies[10,11] have shown that, in between 14% and 16.5% of patients over age 50 with presumed TNP from vascular aetiologies, TNP was actually due to other causes (compressive lesions, aneurysms, demyelinating disease, etc.), and neuroimaging (CT or most recently MRI and MRA) led to an earlier appropriate management of the underlying cause. However, the low cost-benefit ratio of MRI imaging for all patients can be a limiting factor. Thus, some clinicians may use CT scan and

computed tomography angiography (CTA), which are less costly than MRI but may still detect vascular and compressive lesions.[12] In the present case, the physician's confidence in the likelihood of an ischemic cause lead to CT imaging, likely motivated by cost considerations in a publicly funded context. Careful practitioners should therefore consider ordering MRI (with MRA) for all TNP patients, even when an ischemic aetiology is presumed, as well as for cases resulting from suspected compressive or traumatic causes (pupil involvement [anisocoria greater than 2mm], incomplete motility deficit or under age 50).[9–11]

Implications of optometric practice in a prison setting

Optometry in a prison environment is unfamiliar to many practitioners. In Canada, the CSC contracts optometrists as part of their duty of care to offenders, to maintain ocular health and facilitate daily tasks, education and rehabilitation programs and ultimately social reinsertion. This practice has distinct challenges and advantages.

The prison population is underserved, with a significant burden of disease. High levels of systemic disease and risk factors (diabetes, hypertension, hypercholesterolemia, drug and alcohol use, tuberculosis, hepatitis C, etc.) lead to important amounts of diabetic complications, optic neuropathies, cataracts, retinopathies, etc. Referrals to external specialties (e.g. ophthalmology, neuroimaging) may be subject to limitations (availability of transport, security escorts, etc.). Many offenders are not keen to undertake external trips, since transport conditions involve restraints and uncomfortable vehicles. Since an offender may decline medical care (barring specific exceptions) and outside referrals, disease may progress, potentially increasing morbidity. Due to limited budgetary allowances, an institution of 400 to 500 offenders may have access to one day of optometry clinic per month. The number of patients seen in a day (approximately 6 to 14) is in inverse proportion to the security level of an institution. These factors lead to waiting times of 3-12 months for optometric services.

CSC policies for offenders' access to optometric care are outlined in the CSC's National Essential Health Services Framework.[13] Currently, offenders are allowed one eye examination every 2 years, and spectacle replacement (single vision or bifocal lenses) paid by the CSC every 3 years. Eye examination or follow-up visits can be more frequent upon request by the institution's physician or optometrist, as in this case. Due to the great demand and infrequent supply of optometric services, follow-up of certain conditions is more difficult and results in delaying the monitoring of certain conditions, such as, in this case, the patient's opacification of the left posterior capsule.

The CSC provides examination rooms and ophthalmic equipment. Although equipment rooms are meant to enable an optometrist to perform complete eye examinations, in the author's experience equipment is not distributed equally across institutions. Although basic requirements are usually present (ophthalmic chair, phoropter, autorefractor, slit lamp [with fundus and gonioscopy lenses], tonometer, handheld diagnostic set, trial lens set, binocular vision tests, binocular indirect ophthalmoscope, etc.), imaging equipment (fundus photography and optical coherence tomography [OCT]) is conspicuously absent from institutions. Few institutions have automated visual fields. Although not possible in this case, a visual field assessment would have been beneficial, since a compressive lesion along the visual pathway may be highlighted by various types of scotomata. Requests for additional equipment are possible and may be granted, but the decision rests with the CSC authorities according to budgetary allowances and other competing health specialities. If needed, external referrals may be made to local optometric or ophthalmological practices, although these are subject to the same limitations regarding the availability of transport and security escorts.

The clinical environment of a prison also brings distinct advantages to the optometric clinician. The patient's optometric record is part of the offender's complete health record, which includes medical, pharmaceutical, dental, psychosocial, psychiatric and nutritional charts. This provides privileged access to complementary information and test results that greatly contribute to quality optometric care. Furthermore, the health staff of the facilities consists of nurses, who have enlarged roles similar to nurse practitioners, and who are present every day and often overnight. They are key collaborators in delivering comprehensive care to the patients. The medical doctor at each facility, as well as other professionals (pharmacists, nutritionists), is also accessible for consultation. This accessibility is well illustrated in this case, since co-management by the optometrist and the MD led to neuroimaging, the permanence of the nursing staff allowed daily monitoring of pupil function and many professionals were involved in attempting to improve the patient's diabetes control. ●

Acknowledgements

The author would like to thank the Correctional Service of Canada for granting access to the patient's record and to the multidisciplinary teams of dedicated professionals who help to improve and maintain the health of offenders.

References

1. Akagi T, Miyamoto K, Kashii S, Yoshimura N. Cause and prognosis of neurologically isolated third, fourth, or sixth cranial nerve dysfunction in cases of oculomotor palsy. *Jpn J Ophthalmol* 2008; 52: 32–5.
2. Bruce B, Biousse V, Newman N. Third Nerve Palsies. *Semin Neurol* 2007; 27: 257–68.
3. Yanovitch T, Buckley E. Diagnosis and management of third nerve palsy: *Curr Opin Ophthalmol* 2007; 18: 373–8.
4. von Noorden GK, Campos EC. Binocular Vision and Ocular Motility: Theory and Management of Strabismus, 6th Edition. Available at: http://www.cybersight.org/bins/content_page.asp?cid=1-2193. (Accessed: 11th March 2016)
5. Jung JS, Kim DH. Risk factors and prognosis of isolated ischemic third, fourth, or sixth cranial nerve palsies in the Korean population. *J Neuroophthalmol* 2015; 35: 37–40.
6. Lo C-P, Huang C-F, Hsu C-C, *et al.* Neuroimaging of isolated and non-isolated third nerve palsies. *Br J Radiol* 2012; 85: 460–7.
7. Volpe NJ, Lee AG. Do patients with neurologically isolated ocular motor cranial nerve palsies require prompt neuroimaging?: *J Neuroophthalmol* 2014; 34: 301–5.
8. Tamhankar MA, Volpe NJ. Management of acute cranial nerve 3, 4 and 6 palsies: role of neuroimaging. *Curr Opin Ophthalmol* 2015; 26: 464–8.
9. Murchison AP, Gilbert ME, Savino PJ. Neuroimaging and acute ocular motor mononeuropathies: A prospective study. *Arch Ophthalmol* 2011; 129: 301–5.
10. Tamhankar MA, Biousse V, Ying GS, *et al.* Isolated third, fourth, and sixth cranial nerve palsies from presumed microvascular versus other causes: a prospective study. *Ophthalmology* 2013; 120: 2264–9.
11. Chou KL, Galetta SL, Liu GT, *et al.* Acute ocular motor mononeuropathies: prospective study of the roles of neuroimaging and clinical assessment. *J Neurol Sci* 2004; 219: 35–9.
12. Mehta S, Loevner LA, Mikityansky I, *et al.* The diagnostic and economic yield of neuroimaging in neuro-ophthalmology. *J Neuroophthalmol* 2012; 32: 139–44.
13. Correctional Service of Canada. National Essential Health Services Framework. *National Essential Health Services Framework* (2013). Available at: https://buyandsell.gc.ca/cds/public/2014/02/06/98ad3da3e11ddf8a9f779fe07662329e/natessential_hsframework_april20131.pdf. (Accessed: 18th March 2016)

NEW Preservative-Free OTC Eye Drops in a Multi-Dose Bottle

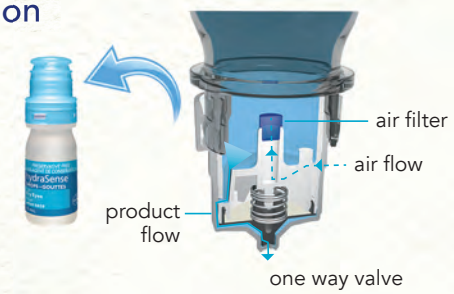
hydraSense®
The Art of **LONG-LASTING RELIEF** FOR DRY EYES

Contains naturally-sourced sodium hyaluronate for long-lasting hydration

- Prolongs tear break-up time and tear film stability[1,2,3,4]
- Has been clinically proven to hydrate the eyes by binding water to protect the cornea from dryness[5,6,7]

Innovative delivery system for a preservative-free solution

- Built-in air filter protects solution from airborne microorganisms, ensuring sterility
- Multi-dose bottle features a one-way valve to prevent contamination by dispensing one sterile drop at a time



Phosphate-free

Can be used with contact lenses

hydraSense®
EYE DROPS

See what hydraSense® Eye Care can do for your patients.

Products available at your local Drug, Mass and Food retailers.

Visit **hydrasense.ca/HCP**

References: 1. Long term treatment with sodium hyaluronate-containing artificial tears reduces ocular surface damage in patients with dry eyes. *Br J Ophthalmol* 2002; 86: 181-184. **2.** Cytoprotective effects of Hyaluronic acid and carbomer 934P in ocular surface epithelial cells. *IOVS*, November 2002, Vol 43, no 11. **3.** Immediate effect of 3% Diquafosol and 0.1% Hyaluronic acid ophthalmic solution on tear break-up time in normal human eyes. *J Ocul Pharmacol Ther.* 2015 Dec; 31 (10): 631-5. **4.** Sustained effects of sodium hyaluronate solution on tear film stability evaluated by Tear Stability Analysis System. *Nippon Ganka Gakkai Zasshi.* 2011 Feb; 115 (2): 134-41. **5.** Characterization of water retentive properties of Hyaluronan. Nakamura et al. *Cornea* 12(5), 433-436, 1993. **6.** Evaluation of sodium hyaluronate as viscous vehicle for eye drops. *J Pharm Belg* 1989, 44, 6: 391-397. **7.** In vitro efficacy of ocular surface lubricants against dehydration. *Cornea* 2013 Sep; 32 (9): 1260-4.

L.CA.MKT.CC.03.2017.0659

BAYER

Gestion optométrique d'une paralysie du troisième nerf crânien dans un milieu carcéral

Benoît Tousignant, OD, M. Sc., MPH, FAAO
Professeur adjoint
École d'optométrie,
Université de Montréal

Résumé

La paralysie du troisième nerf crânien (PTN) est un trouble neuro-ophtalmique que les optométristes peuvent rencontrer dans divers contextes cliniques. Elle affecte les muscles extraoculaires et peut nuire à divers degrés aux fonctions de la paupière et de la pupille. Chez les adultes, son étiologie est souvent associée à des lésions par compression du système nerveux central ou à des causes d'ischémie vasculaire découlant de maladies systémiques comme le diabète et l'hypertension. Le présent rapport de cas s'intéresse à la gestion d'une PTN dans un milieu carcéral canadien multidisciplinaire, milieu dans lequel la population de patients est souvent mal desservie. Il présente les pratiques actuelles en matière de neuro-imagerie pour les cas de PTN dans un contexte clinique conventionnel. Il aborde également les enjeux et avantages de la gestion de la PTN dans un milieu carcéral, en soulignant les particularités des soins aux patients vulnérables en milieu carcéral.

MOTS CLÉS
Paralysie du troisième nerf crânien, pénitencier, soins interdisciplinaires, neuro-imagerie, population mal desservie

INTRODUCTION

La paralysie du troisième nerf crânien affecte les muscles extraoculaires, la paupière et la pupille et est un trouble rencontré par beaucoup d'optométristes. Le présent rapport de cas souligne les particularités de la gestion de ce trouble dans un milieu carcéral multidisciplinaire.

RAPPORT DE CAS

Un homme caucasien de 60 ans s'est présenté au service d'optométrie d'un pénitencier canadien de sécurité moyenne après avoir été aiguillé par le médecin de l'unité de santé mentale du pénitencier en raison d'une « vision réduite ou double ».

En réponse aux questions, le patient s'est surtout plaint d'avoir une vision floue depuis qu'il a perdu s'est lunettes. Il a nié la présence de diplopie, de douleur, de maux de tête et de traumatisme oculaire. Les réponses du patient étaient quelque peu limitées en raison de ses problèmes de santé mentale et d'un défaut de prononciation. Il a subi une ablation des cataractes en 2009 et porte habituellement des lunettes pour corriger un astigmatisme hypermétropique composé depuis 2011. La vision binoculaire et la santé oculaire ne présentaient par ailleurs aucune particularité.

Son historique médical actuel incluait le diabète de type 2 (1999, récente HbA1c 10,1 %), l'obésité, l'hypertension, l'hypercholestérolémie, l'hyperplasie prostatique bénigne, la neuropathie diabétique, l'apnée du sommeil, le bégaiement, l'anémie, une légère déficience intellectuelle et la schizophrénie paranoïde. Il était traité avec de la gabapentine, de l'olanzapine, du valproate, de l'atorvastatine, de l'ézétimibe, du furosémide, de l'irbésartan, de la nifédipine, de l'insuline, du sulfate ferreux et de la térazosine. Ni l'historique familial médical ni l'historique familial oculaire n'étaient disponibles.

L'examen externe a révélé une ptose complète à l'œil droit, qui n'avait pas été mentionnée par le patient ni le médecin. Les acuités visuelles de loin non corrigées étaient de 6/21 OD (en tenant la paupière ouverte) et de 6/12 OS, avec un test de sténopé peu concluant. La distance manifeste de réfraction a démontré une ordonnance hypermétropique composée stable, avec des acuités visuelles corrigées de 6/6 OD (en tenant la paupière ouverte) et de 6/7,5 OS. Le test sous écran était peu concluant (compréhension et fixation difficiles), mais l'alignement oculaire par les réflexes de Krimsky a démontré un dioptre d'exotropie de 30 prismes et un dioptre d'hypotrophie de 10 prismes à l'œil droit. L'œil droit montrait des limitations de grade -4 pour l'adduction, l'abaissement et l'élévation, tandis l'œil gauche montrait une amplitude complète de la mobilité dans toutes les directions. Les pupilles étaient égales, réagissaient à la lumière et avaient une accommodation, sans déficit pupillaire afférent. L'examen du segment antérieur a révélé une ptose complète à l'œil droit, mais aucune autre particularité. La pression intraoculaire était de 14 mmHg dans les deux yeux. La lentille intraoculaire gauche avait une légère opacification de la capsule postérieure. L'examen du fond de l'œil sous mydriatique n'a révélé aucune preuve de rétinopathie diabétique. Le champ visuel automatisé n'était pas disponible, et les champs visuels par confrontation étaient peu fiables.

Ce portrait clinique suggère une PTN complète, isolée avec une pupille fixe, à la lumière des éléments suivants : unilatérale, indolore, atraumatique, apparition récente d'une exophorie et hypotrophie, ptose complète, patient âgé de plus de 50 ans, fonction pupillaire normale et fonction normale des nerfs crâniens quatre et six. Le diabète est considéré être la cause la plus probable, compte tenu du profile glycémique du patient et de son hypertension systémique qui est sous contrôle. Le médecin du pénitencier a été consulté, et il a demandé une consultation en neuro-imagerie afin d'exclure la possibilité d'étiologies par compression. Le personnel infirmier a été avisé de surveiller le patient quoditiennement pendant la semaine suivante pour détecter tout développement tardif d'une dilatation de la pupille droite. Un rendez-vous de suivi en optométrie a été fixé trois semaines plus tard. De nouvelles lunettes ont été commandées, mais aucune gestion de diplopie n'a été jugée nécessaire en raison de la ptose complète. Le patient a reçu des conseils quant à l'importance du contrôl de la glycémie et aux effets des maladies oculaires imputables au diabète. Il a été demandé à la nutritionniste du pénitencier d'intervenir afin d'insister sur les recommandations alimentaires. À l'examen de suivi, le patient était plus alerte, ce qui a facilité l'anamnèse. En réponse aux questions, il a nié avoir de la diplopie et de la douleur et semblait heureux de la nouvelle correction de la vue. Les résultats de neuro-imagerie n'étaient pas encore disponibles. Le dossier du patient indiquait qu'il n'y avait pas de changements de fonction de pupille. Les acuités visuelles corrigées étaient 6/6 OD et de 6/7,5 OD. L'examen externe a démontré qu'il n'y avait pas de ptose. Les yeux du patient étaient maintenant alignés lors de la fixation primaire du regard, avec un dioptre d'exotropie de 30 prismes de loin et de près, une vision binoculaire normale (filtre rouge) et une stéréopsie. Les mobilités extraoculaires avaient une amplitude complète dans toutes les directions. Les réflexes pupillaires étaient normaux, sans anisocorie. Un examen de suivi a été planifié deux mois plus tard. Le patient a été

avisé d'alerter le personnel infirmier de toute diplopie ou tout changement dans sa vision ou apparence de sa pupille ou de sa paupière.

À l'examen de suivi deux mois plus tard, le patient a déclaré n'avoir observé aucun changement de son état. Les acuités visuelles corrigées étaient stables, et l'examen externe était normal. Le patient présentait maintenant une orthophorie de loin et de près. Les mobilités extraoculaires avaient une amplitude complète dans toutes les directions, et les réflexes pupillaires étaient normaux. Le rapport de radiologie incluait une tomodensitométrie de la tête et des orbites, avec contraste, qui montrait qu'il n'y avait pas de lésions intracrâniennes ou d'anomalies significatives. Le patient a été informé de son rétablissement complet en apparence et a été avisé de revenir dans deux ans, conformément à la politique du Service correctionnel du Canada à propos de la fréquence des examens ophtalmologiques.

DISCUSSION

Présentation clinique et pathophysiologie

Le troisième nerf crânien innerve les muscles supérieur, inférieur et droits internes, de même que l'oblique inférieur et releveur de la paupière. Ses fibres externes et dorsolatérales contrôlent l'accommodation et la myosis. Ainsi, la PTN se présente habituellement avec une ptose ipsilatérale, une hypotrophie avec exotropie concomitante, diplopie, et une possible pupille fixe et mydriatique, et une vision floue[1, 2]. Une PTN complète inclut l'incapacité à à élever, baisser ou d'exercer une adduction de l'œil et est accompagnée d'une ptose complète. L'implication de la pupille dépend de l'étiologie. Une PTN complète ou partielle restreindra à divers degrés les mobilités extraoculaires en compromettant un ou plusieurs muscles extraoculaires et cause des implications pupillaires variables. Les paralysies partielles affectent le plus souvent soit la branche inférieure du nerf (muscle droit médial de l'œil, muscle droit inférieur de l'œil et le muscule oblique inférieur) ou sa branche supérieure (muscle droit supérieur de l'œil et le releveur).[1,3] La plupart des

cas de PTN sont unilatéraux – les cas bilatéraux ont lieu seulement lorsque le noyau oculomoteur est impliqué.

Les étiologies principales des PTN incluent les causes vasculaires ou ischémiques (diabète, hypertension, maladie athérosclérotique), la compression (anévrisme, tumeur), les traumatismes et les causes congénitales[1, 3, 4]. L'étiologie est déterminée par les facteurs qui incluent l'implication de la pupille, la douleur, les facteurs de risque vasculaires et l'âge. Habituellement, la mydriase indiquera une lésion par compression[1, 3]. Le nerf chemine du mésencéphale à l'orbite par l'espace sous-arachnoïdien, il passe à côté du polygone de Willis, près de l'artère communicante postérieure, là où un anévrisme peut compresser les fibres externes pupillaires, entraînant une mydriase et une perte de la fonction pupillaire. Les paralysies par compression sont souvent accompagnées de douleur ou maux de tête[3, 4]. Inversement, les causes ischémiques n'affecteront généralement que les fibres internes du troisième nerf crânien résultant en une pupille fixe indolore[4, 5]. Chez les patients âgés de plus de 50 ans, il est plus probable que la PTN soit de nature ischémique, où le *vasa nervorum* peut souffrir d'ischémie, affectant ainsi les fibres internes qu'il alimente. Les patients de moins de 50 ans sont plus susceptibles de souffrir d'étiologies compressives[1]. La PTN peut être combinée à des paralysies du quatrième, cinquième ou sixième nerfs crâniens avec des lésions affectant le sinus caverneux[6].

Diagnostic différentiel

Dans le présent cas, le diagnostic différentiel inclut la myasthénie grave, la PTN partielle et une déviation pré-existante ou décompensée avec une ptose concomitante. Ce dernier a été exclu sur la base des dossiers médicaux antérieurs qui montraient un alignement oculaire normal et une fonction normale de la paupière. Une paralysie partielle a été exclue compte tenu de l'handicap complet de la paupière et des muscles extraoculaires. Pour terminer, une myasthénie oculaire grave peut être présente avec de la diplopie et toucher les muscles extraoculaires ou les muscles releveurs. Cependant, les déséquilibres musculaires augmenteront généralement avec la fatigue musculaire. Cela a été exclu dans le cas présent puisque la ptose était complète et constante, l'adduction et l'élévation étant complètement altérées.

Gestion du cas

La période de rétablissement pour une PTN dont les causes suspectées sont de nature vasculaire est habituellement entre trois et 12 mois[1, 3]. Sa gestion implique de surveiller les symptômes de diplopie, de surveiller l'implication pupillaire, de réaliser une neuro-imagerie etde cibler la condition systémique sous-jacente[3, 4]. La gestion de la dip-

lopie exige l'application d'un timbre ou l'utilisation de lentilles givrées ou de prismes de Fresnel pour réduire les symptômes[3, 4]. Si les prismes de Fresnel sont simples et abordables, les lentilles givrées sont coûteuses et possèdent de longs délais de livraison, alors qu'il s'agit probablement d'une solution temporaire. Dans le présent cas, bien que l'anamnèse était peu fiable, la présence d'une ptose complète permettait d'éliminer des affections potentielles de diplopie. De plus, lorsque la ptose s'est atténuée, le patient a également retrouvé une vision binoculaire.

Comme certaines causes compressives se présentent initialement avec une fonction pupillaire normale, une pupille fixe d'une PTN ischémique présumée doit être suivie de près pendant la première semaine pour vérifier l'apparition d'implication pupillaire[1, 3, 6]. Dans le présent cas, la surveillance a été assurée par le personnel infirmier qui était présent tous les jours.

Rôle de la neuro-imagerie

Les données probantes fondées sur la neuro-imagerie quant à la PTN ischémique présumée chez les patients de plus de 50 ans a évolué au fil du temps et demeure controversée[7, 8]. Le problème principal concerne la valeur diagnostique de la neuro-imagerie pour éliminer l'anévrisme ou d'autres lésions par compression comme causes de tels cas. Les pratiques actuelles en ophtalmologie ont évolué de l'observation minutieuse et attentive (de 3 à 6 mois) à la réalisation d'imagerie par résonnance magnétique (IRM) et d'angiographie par résonnance magnétique (ARM) pour la plupart des patients ayant une PTN, de manière à éliminer le plus efficacement la possibilité de lésions par compression[6, 9-11]. Malgré un manque actuel de données probantes de haute qualité (revue systématique, méta-analyses ou directives cliniques fondées sur les données cliniques) à ce sujet, les études prospectives[10, 11] ont démontré que, dans 14 % à 16,5 % des cas de patients de plus de 50 ans ayant une PTN qui était présumée avoir des étiologies vasculaires, les causes de la PTN étaient autres en réalité (lésions par compression, anévrismes, maladie démyélinisante, etc.), et que la neuro-imagerie (tomodensitométrie et plus récemment IRM et ARM) a mené à une gestion appropriée plus rapide de la cause sous-jacente. Cependant, le faible ratio coût-bénéfice de la neuro-imagerie par IRM pour tous les patients peut être un facteur limitant. Par conséquent, certains cliniciens peuvent utiliser la tomodensitométrie et l'angiographie par tomodensitométrie (AT) qui sont moins dispendieuses que l'IRM, mais qui peuvent tout de même détecter les lésions par compression et les lésions vasculaires[12]. Dans le cas présent, la conviction du médecin quant à la probabilité d'une cause ischémique a mené à une tomodensitométrie, vraisemblablement motivée par des considérations de coût dans un contexte de soins subventionnés par le public. Des praticiens attentionnés devraient donc demander des IRM (avec ARM) pour tous les patients ayant une PTN, même si l'étiologie ischémique est présumée, de même que pour les cas susceptibles d'être causés par compression ou par traumatisme (implication pupillaire [anisocorie supérieure à 2 mm], déficit partiel de la mobilité ou mois de 50 ans)[9-11].

Implications de la pratique optométrique en milieu carcéral

Beaucoup de praticiens connaissent mal la pratique optométrique en milieu carcéral. Au Canada, le SCC engage des optométristes dans le cadre de leurs obligations de soins aux contrevenants, en vue de préserver la santé oculaire, de faciliter les tâches quotidiennes, de favoriser les programmes d'éducation et de réhabilitation et ultimement la réinsertion sociale. Cette pratique a des enjeux et des avantages distincts.

La population en milieu carcéral est mal desservie, avec un fardeau significatif de maladies. Les niveaux élevés de maladie systémique et les facteurs de risque (p. ex. diabète, hypertension, hypercholestérolémie, utilisation de drogue et d'alcool, tuberculose, hépatite C, etc.) mènent à un nombre significatif de complications du diabète, neuropathie optique, cataractes, rétinopathies, etc. Les aiguillages vers des spécialistes à l'externe (p. ex. ophtalmologie, neuro-imagerie) peuvent être soumis à des limitations (disponibilité du transport, escortes de sécurité, etc.). Beaucoup de contrevenants ne désirent pas faire des voyages à l'extérieur du milieu carcéral puisque les conditions de transport impliquent des contraintes et des véhicules qui ne sont pas confortables. Le refus par un contrevenant de recevoir des soins médicaux (sauf lors d'exceptions spécifiques) et d'être référé à l'externe peut se traduire par la progression d'une maladie ce qui potentiellement peut augmenter la morbidité. En raison de dotations budgétaires limitées, un établissement de 400 à 500 contrevenants peut avoir accès à une clinique d'optométrie une journée par mois. Le nombre de patients vus en une journée (approximativement 6 à 14) est inversement proportionnel au niveau de sécurité de l'établissement. Ces facteurs mènent à des temps d'attente de 3 à 12 mois pour avoir accès à des services optométriques.

Les politiques du SCC relativement à l'accès des contrevenants aux soins optométriques sont énoncés dans Cadre national des services de santé essentiels du SCC[13]. À l'heure actuelle, les contrevenants sont autorisés à avoir un examen de la vue tous les deux ans et à avoir un remplacement de leurs lunettes (verres simples ou lunettes bifocales) payé par le

SCC tous les trois ans. L'examen de la vue et les visites de suivi peuvent être plus fréquentes sur demande du médecin ou de l'optométriste de l'établissement, comme dans le cas présent. En raison de la demande importante et l'offre ponctuelle de services optométriques, le suivi de certaines conditions est plus difficile et engendre des retards dans la surveillance de certaines troubles, comme l'opacification de la capsule postérieure gauche du patient dans le cas présenté

Le SCC fournit des salles d'examen et de l'équipement ophtalmique. Bien que les salles d'examen sont destinées à permettre à un optométriste à exécuter des examens complets des yeux, l'auteur du présent texte a constaté par son expérience que l'équipement n'est pas distribué de manière égale entre les établissements. Bien que les exigences de base soient habituellement présentes (chaise ophtalmique, réfracteur, autoréfractomètre, lampe à fente [avec lentille de fond et le goniomètre à prisme], tonomètre, trousse de diagnostic de poche, trousse de lentilles d'essais, tests de vision binoculaire, ophtalmoscope indirect, etc.), l'équipement d'imagerie (photographie de fond et tomographie par cohérence optique [TCO]) manque cruellement dans les établissements. Quelques établissements ont des champs visuels automatisés. Bien que ce n'était pas possible dans le présent cas, une évaluation du champ visuel aurait été bénéfique, car une lésion par compression située le long de la voie optique peut être soulignée par divers types de scotomes. Des requêtes pour de l'équipement additionnel sont possibles et peuvent être acceptées, mais la décision repose entre les mains des autorités du SCC et est prise selon les dotations budgétaires et autres spécialités médicales. Au besoin, les consultations à l'externe peuvent être réalisées auprès d'un optométriste ou ophtalmologiste de la région, bien que ces requêtes soient assujetties aux mêmes contraintes concernant la disponibilité du transport et les escortes de sécurité.

L'environnement clinique dans le milieu carcéral apporte également des avantages distincts au clinicien en optométrie. Le dossier optométrique du patient est une partie du dossier médical complet du contrevenant, lequel inclut des fiches médicales, pharmaceutiques, dentaires, psychosociales, psychiatriques et nutritionnelles. Cela fournit un accès privilégié à de l'information complémentaire et à des résultats de test qui contribuent grandement à la qualité des soins optométriques. De plus, le personnel des services de santé des établissements est composé d'infirmières, qui ont des rôles élargis similaires à ceux des infirmières praticiennes, et est présent chaque jour et est souvent présent pendant la nuit. Ces infirmières sont des collaboratrices clés qui fournissent des soins complets aux patients. Les médecins de chaque établissement, de même que d'autres professionnels (pharmaciens, nutritionnistes) sont également disponibles pour la consultation. Cette accessibilité est bien illustrée dans le cas présent, car la co-gestion qui a été faite par l'optométriste et le médecin a mené à l'imagerie et la permanence du personnel infirmier a permis une surveillance quotidienne de la fonction pupillaire et plusieurs professionnels ont été impliqué à tenter de contrôler le diabète du patient. ●

Remerciements

L'auteur tient à remercier le Service correctionnel du Canada d'avoir autorisé l'accès au dossier du patient et aux équipes multidisciplinaires composées de professionnels dévoués qui aident à améliorer et préserver la santé des contrevenants.

Références

1. Akagi, T., K. Miyamoto, S. Kashii et N. Yoshimura. « Cause and prognosis of neurologically isolated third, fourth, or sixth cranial nerve dysfunction in cases of oculomotor palsy », *Jpn J Ophthalmol,* 2008; nº 52, p. 32-35.
2. Bruce, B., V. Biousse et N. Newman. « Third Nerve Palsies », *Semin Neurol,* 2007; nº 27, p. 257-268.
3. Yanovitch, T., et E. Buckley. « Diagnosis and management of third nerve palsy », *Curr Opin Ophthalmol,* 2007; nº 18, p. 373-378.
4. von Noorden, G.K., et E.C. Campos. *Binocular Vision and Ocular Motility: Theory and Management of Strabismus*, 6e édition. En ligne : http://www.cybersight.org/bins/content_page.asp?cid=1-2193 (consulté le 11 mars 2016).
5. Jung, J.S., et D.H. Kim. « Risk factors and prognosis of isolated ischemic third, fourth, or sixth cranial nerve palsies in the Korean population », *J Neuroophthalmol*, 2015; nº 35, p. 37-40.
6. Lo, C.-P., C.-F. Huang, C.-C. Hsu et coll. « Neuroimaging of isolated and non-isolated third nerve palsies », *Br J Radiol,* 2012; nº 85, p. 460-467.
7. Volpe, N.J., et A.G. Lee. « Do patients with neurologically isolated ocular motor cranial nerve palsies require prompt neuroimaging? », *J Neuroophthalmol,* 2014; nº 34, p. 301-305.
8. Tamhankar, M.A., et N.J. Volpe. « Management of acute cranial nerve 3, 4 and 6 palsies: role of neuroimaging », *Curr Opin Ophthalmol*, 2015; nº 26, p. 464-468.
9. Murchison, A.P., M.E. Gilbert et P.J. Savino. « Neuroimaging and acute ocular motor mononeuropathies: A prospective study », *Arch Ophthalmol*, 2011; nº 129, p. 301-305.
10. Tamhankar, M.A., V. Biousse, G.S. Ying et coll. « Isolated third, fourth, and sixth cranial nerve palsies from presumed microvascular versus other causes: a prospective study », *Ophthalmology*, 2013; nº 120, p. 2264-2269.
11. Chou, K.L., S.L. Galetta, G.T Liu et coll. « Acute ocular motor mononeuropathies: prospective study of the roles of neuroimaging and clinical assessment », *J Neurol Sci*, 2004; nº 219, p. 35-39.
12. Mehta, S., L.A. Loevner, I. Mikityansky et coll. « The diagnostic and economic yield of neuroimaging in neuro-ophthalmology », *J Neuroophthalmol,* 2012; nº 32, p. 139-144.
13. Service correctionnel du Canada. *Cadre national relatif aux soins de santé essentiels*, 2015. En ligne : https://buyandsell.gc.ca/cds/public/2014/02/06/98ad3da3e11ddf8a9f779fe07662329e/natessential_hs-framework_april20131.pdf (consulté le 18 mars 2016).

CANADIAN CERTIFIED
OPTOMETRIC ASSISTANT
(CCOA) PROGRAM

The staff you've always wanted already work for you.

+ Invest in your staff – improve your practice.

Our new and improved Canadian Certified Optometric Assistant (CCOA) online distance education program gives optometric assistants the skills and knowledge they need to contribute to a thriving optometric practice.

REGISTER NOW!

New session begins **September 15**.
Registration **August 21 - September 8/$1875**

Learn more at
www.opto.ca/ccoa

CANADIAN ASSOCIATION OF OPTOMETRISTS
ASSOCIATION CANADIENNE DES OPTOMÉTRISTES

We hire doctors who want to be doctors.

Your patients won't care about your skills in finance, marketing or HR.

Build your career on a passion for eye care and a reputation as an exceptional optometrist and you'll go far.

Join forces with us.

FYidoctors enjoy current diagnostic equipment, buying power and access to our Canadian lens lab.

They also have expert resources on-demand to help them grow a profitable business, so they can focus on what they're great at.

We put patients first. It's why we're Canada's largest partnership of optometrists, coast to coast.

fyidoctors.com

FYidoctors™

For you. For your eyes.

Idiopathic Juxtafoveal Telangiectasia

Jessica Haynes, OD, FAAO

Jim Williamson, OD, FAAO

Abstract

Idiopathic Juxtafoveal Telangiectasia (IJT), also called Idiopathic Macular Telangiectasia, is a group of disorders with varying etiologies that share the common finding of telangiectatic vessels in the juxtafoveal region of the macula. IJT is classically divided into three groups, each with clinically distinct findings and differing etiologies. Type I typically occurs unilaterally, primarily affects males, and is thought to be congenital. Vision loss usually results from macular edema. The most common type, II, normally presents bilaterally, has no sex predilection, and is thought to be acquired. Vision loss occurs not from edema, but from retinal atrophy. The last type, III, is extremely rare and is usually found in association with coexisting systemic or neurological disorders. Vision loss most likely results from retinal ischemia. This paper will describe classifications, clinical findings, etiologies, and treatment options of IJT along with patient case figures.

KEY WORDS:
Idiopathic Juxtafoveal Telangiectasia, Idiopathic Macular Telangiectasia

INTRODUCTION

IJT is a condition comprised of three classical divisions, each with different clinical findings, etiologies, and patient demographics. They do share a common feature of capillary abnormalities in the juxtafoveal region that appear on fluorescein angiography as dilated or telangiectatic capillary changes.[1] IJT can easily be confused for other conditions, and these abnormal capillary findings must be separated from those resultant from other conditions such as diabetes, carotid occlusive disease, retinal vein occlusion, radiation therapy, and others.[2] The classification also does not include Coat's disease, which has more widespread retinal telangiectatic changes. However, Coat's disease and IJT Type I could be subsets of the same condition that exists as a spectrum of disease.[3]

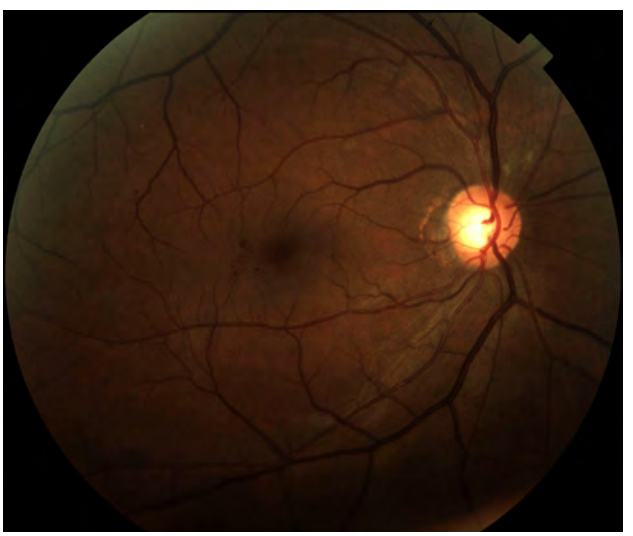
GASS AND BLODI CLASSIFICATION

Gass and Owakawa first coined the term IJT in 1982 and initially categorized the condition in four different classes.[1] Gass and Blodi later revisited the topic in 1993 in a follow up report of the initial work done by Gass and Owakawa at which time they modified the classification into three main categories, each with separate unrelated etiologies and two subsets: Type IA and B, Type IIA and B, and Type IIIA and B. Additionally, Type II was further divided into five stages.[3] Clinical observations and fluorescein angiography (FA) findings formed the basis of the grouping in this system.

Type I

Type I IJT presented with easily visible retinal telangiectasia often accompanied with exudation and macular edema, with vision loss resulting from the latter. Gass and Blodi broke down Type I IJT based on the extent of telangiectasia as defined by the number of involved clock hours around the fovea. Type IA had more than two clock hours of telangiectasia while Type IB had less than two clock hours (Figure 1).

Figure 1: *Easily visible retinal telangiectasia in a patient with Gass and Blodi Type IA IJT, or Yanuzzi et al.'s Type I aneurysmal telangiectasia.*



In Gass and Blodi's study, Type IA patients consisted of 31 subjects with a high male predilection (28 male) and a mean patient age of 37. Entering visual acuity ranged from 20/20 to 20/200. They found Type IA to be unilateral in 30 of the 31 cases. Clinical findings varied between patients, but generally presented with easily visible retinal telangiectasia, macular edema, and exudation. Type IA patients lacked right angle veins, superficial crystalline deposits, intraretinal pigmentary plaques, or subretinal neovascularization (SRNV).

Fluorescein angiography showed quick filling of the irregular telangiectatic vessels around the fovea. These appeared as an irregular round zone of capillary aneurysms and dilated capillaries located temporal to the fovea in 94% of the cases. They noted minimal capillary occlusion or capillary dropout. Late stage FA revealed intraretinal staining as the abnormal vessels continued to leak.

Only eight patients in the study displayed the less prevalent Type IB IJT. Again, a high gender predilection existed with seven of the patients being male. The average age was 42. All but two eyes (20/25 and 20/30) exhibited 20/20 visual acuity.

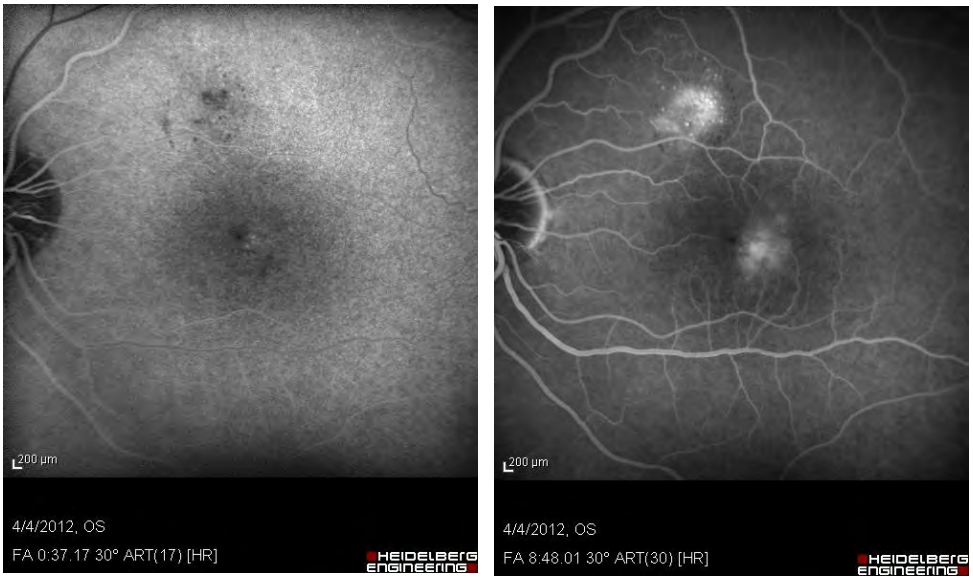
Findings were again unilateral in most cases (seven out of eight) but were confined to two clock hours or less of still easily visible telangiectasia. A few flecks of exudates occurred in most cases, but less than that seen with Type IA. Fluorescein angiography showed early filling of one to four capillary aneurysms and of the irregular capillaries followed by late staining around the rare aneurysms.

Type II

Unlike Type I, Type II IJT rarely displays macular edema and exudation. In these patients, outer retina atrophy is the main cause of vision loss. Gass and Blodi classified Type II into subsets A and B based on the age of presentation.

Type IIA consisted of 92 patients with equal gender incidence and an average age of 55 years. Entering visual acuity ranged from 20/15 to hand motion. Presentation was bilateral, though often asymmetric, in 90 out of 92 patients. Unlike Type I, these patients exhibited either minimal, poorly visible, or no clinically discernible telangiectasia. These abnormal capillaries would light up early with FA and then gradually stain in late stages (Figure 2). All cases involved the temporal parafovea. Additional clinical findings of Type IIA included right angle veins, pigmented retinal plaques, superficial crystalline deposits, and SRNV (Figures 3 and 4). Gass and Blodi also described what appeared to be foveal atrophy on funduscopic evaluation, similar to that of a lamellar macular hole (Figure 4).

Figure 2: *Early phase FA (left) shows the classic finding of punctate hyperfluorescence temporal to the fovea and the atypical location of more areas superior to the macula. Late phase reveals increased hyperfluorescence (right).*



Gass and Blodi developed five stages of Type IIA. In their findings, visual acuity remained unaffected in Stages 1 and 2. Patients typically became symptomatic in Stage 3, and Stage 5 resulted in poor visual acuity.

- Stage 1: Biomicroscopically normal fundus, minimal or no capillary abnormality in early FA, mild staining in late FA
- Stage 2: Slight graying of perifoveal retina, minimal or no visible telangiectasia, capillary telangiectasia in early FA
- Stage 3: Right angle veins
- Stage 4: Pigmented retinal plaques
- Stage 5: Subretinal neovascularization

Figure 3: *Gass and Blodi Type IIA Stage 4 (note the prominent right angle vein stretching from the superior temporal venous branch directly to the pigment plaque), or Yanuzzi et al.'s Type II perifoveal telangiectasia, non-proliferative stage.*

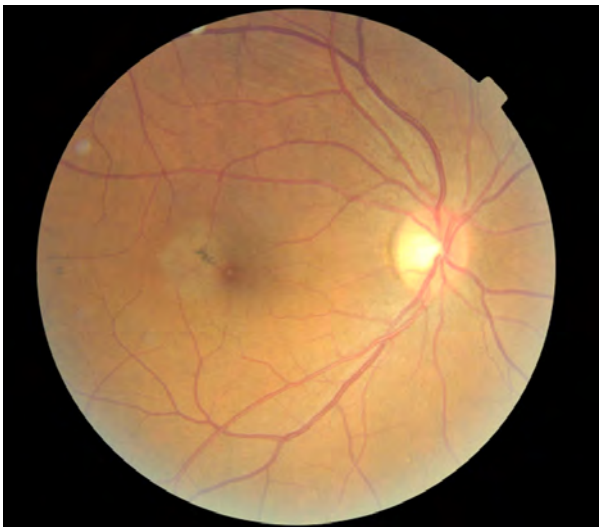
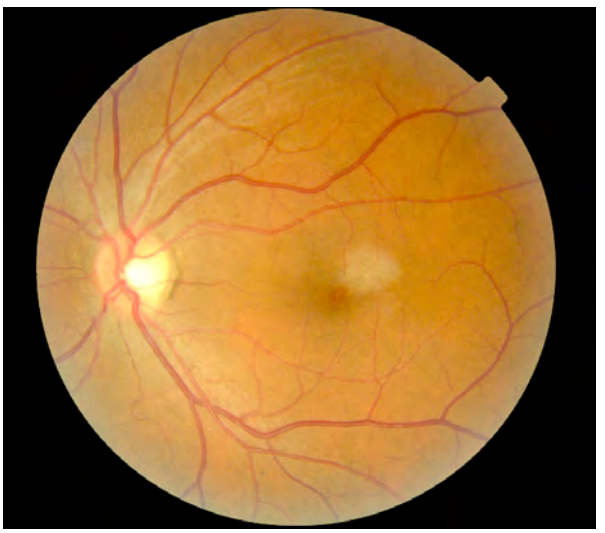


Figure 4: *Type II IJT with crystalline deposits, perifoveal retinal whitening, and a foveal appearance similar to that of a lamellar macular hole.*



Type IIB consisted of only two patients who were brothers aged nine and 12. Both presented with bilateral SRNV and subtle juxtafoveal telangiectasia, but without right angle veins, crystalline deposits, or pigmented plaques.

Type III

Occlusive capillary dropout along with retinal telangiectasia characterized Type III. All had minimal exudation and edema. Type IIIA consisted of three women aged 41 to 59, all with bilateral findings. Their visual acuity ranged from 20/20 to 20/50. One reported polycythemia vera, another gout, and a third hyperglycemia. Type IIIB consisted of five men aged 35 to 41 all with CNS disorders. Two of them were brothers.

YANUZZI ET AL. CLASSIFICATION

In 2006, Yanuzzi et al. proposed a simpler classification based on clinical, FA, and optical coherence tomography (OCT) findings in patients with IJT. Again there were three main categories, but without subsets in each main division. They referred to Type I as "aneurysmal telangiectasia", Type II as "perifoveal telangiectasia", and Type III as "occlusive telangiectasia". Similar to Gass and Blodi, Yanuzzi et al. further divided Type II into stages, but only two instead of five: Non-proliferative and proliferative.[4]

Type I – Aneurysmal Telangiectasia

Yanuzzi et al. reclassified Type IA and IB to "aneurysmal telangiectasia." In this study, those with less than two clock hours of telangiectasia eventually progressed to having more extensive involvement. Therefore, they felt that Type IB was just an earlier presentation of Type IA (Figure 1).

There were 10 patients (nine men, one female) in the report with aneurysmal telangiectasia with an average age of 56. All but the female patient displayed unilateral findings consistent with those reported by Gass and Blodi, such as easily visible telangiectasia, exudation, and macular edema. OCT confirmed the presence of macular edema. Once again there were no findings of neovascularization, pigment proliferation, or crystalline deposits, and there was minimal ischemia.

Type II – Perifoveal Telangiectasia

"Perifoveal telangiectasia" replaced both Type IIA and Type IIB. Yanuzzi et al. believed Type IIB to be a familial abnormality as there had been no other reported cases in the literature. They also found that not all patients went through each stage of Type II previously described by Gass and Blodi, and so they categorized Type II into proliferative and non-proliferative based on the presence or absence of SRNV.

Perifoveal telangiectasia patients in this study consisted of 11 males and 15 females with an average age of 59. Clinical and FA findings were similarly consistent with those seen by Gass and Blodi (Figure 2). All cases were bilateral, but again with some asymmetry. Early signs included mild loss of retinal transparency and mild perifoveal retinal whitening without obvious telangiectasia which appeared later in the disease. Also seen in some were pigment plaques, crystalline deposits, and right angle vessels which Yanuzzi et al. explained could be arteriolar or venular in origin (Figures 3 and 4).

Optical coherence tomography findings provided enlightening insight into perifoveal telangiectasia. Even though the FA showed late staining, OCT confirmed the absence of intraretinal edema. Cystic retinal spaces often bordered anteriorly by the internal limiting membrane (ILM) were present, but they were not secondary to edematous leakage. Instead, they resulted from retinal atrophy. Yanuzzi et al. called these spaces inner lamellar cysts with ILM drape (Figure 5). Optical coherence tomography also revealed a progressive loss of the outer retina along with photoreceptor atrophy (Figure 6). The level of atrophy correlated with the patients' visual acuities. Pigmentary plaques appeared as nonspecific subretinal reflectance with posterior shadowing. The crystalline deposits were too small to image with OCT. In the event of SRNV, resultant macular edema, hemorrhage, macular detachment, or fibrosis could also be visualized with OCT.

Due to the apparent features of Type II IJT seen with OCT imaging, some suggest new grading criteria based on OCT findings.[5,6] In addition to Yanuzzi et al.'s OCT findings, additional reports describe early retinal changes using macular pigment optical density, confocal scanning laser ophthalmoscope, and fundus autofluorescence (FAF).[7] According to some, FAF may detect the earliest signs of Type II IJT due to macular pigment breakdown which allows increased autofluorescence to show through from the underlying retinal pigment epithelium (RPE).[8,9] It eventually affects the RPE itself and causes an increase in both lipofuscin and subsequent autofluorescence (Figure 7). Further release of disrupted RPE pigment causes a blocking effect and leads to a mottled appearance of both hyper- and hypo-fluorescence.[8,9]

Type III – Occlusive Telangiectasia

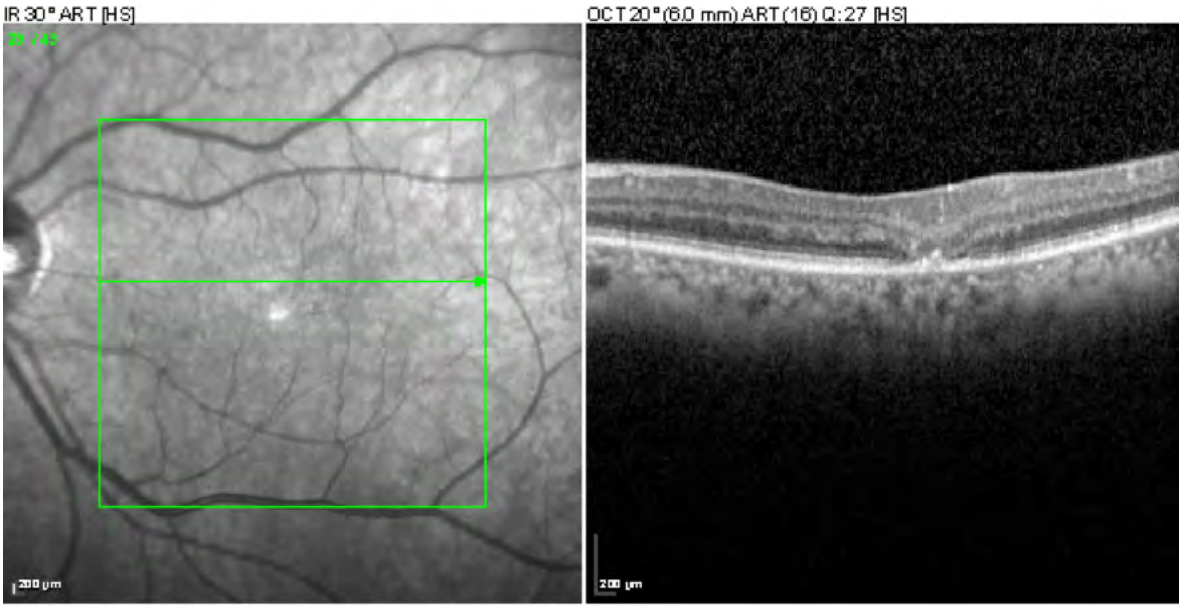
Yanuzzi et al. did not include any Type III or occlusive telangiectasia cases in their report. They believed it to be primarily an ischemic foveal disease with compensatory changes in the capillary bed, and argued that it be omitted from the macular telangiectasia classification as it is an ocular manifestation of systemic or cerebral familial disease.

PROPOSED ETIOLOGIES

Type I IJT is thought to be a congenital malformation of the retinal vasculature which allows for leakage of fluid and exudation from the capillary vessels.[3,4] As mentioned previously, edema is the main cause of vision loss in these patients. Yanuzzi et al. and Gass and Blodi both considered that Type I IJT or aneurysmal telangiectasia could be a more focal variant of Coat's disease and that perhaps they exist on a spectrum of diseases caused by congenital retinal telangiectasia formation.

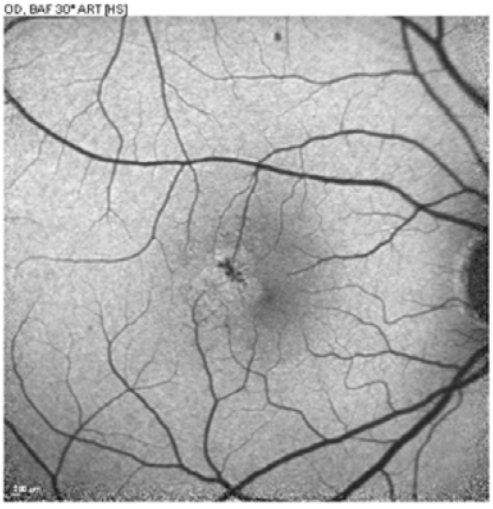
Figure 5: *SD-OCT of an intraretinal cyst with overlying ILM drape and underlying photoreceptor damage.*

Figure 6: *SD-OCT reveals outer retinal atrophy.*



Type II IJT may be an acquired condition in which the malformations of the retinal vasculature present later in life.[3,4] A histological study performed by Green et al. on a patient with Type II IJT showed narrowing of the vessel lumen versus the telangiectatic or dilated appearance noted clinically and on FA. Increased width of the basement membrane along with degeneration of pericytes and endothelial cells led to thickening of the retinal capillaries. The authors conceived that breaks in the endothelium allowed fluorescein to leak into the thickened capillary walls which accounted for the late staining seen on FA. This report also found that capillary proliferation extended into the outer retinal layers down to the photoreceptors.[10] A different histopathological report from a more advanced case of Type II IJT with SRNV did show dilation of retinal capillaries that reached into the outer retinal layers and subretinal space. Pigment migration also occurred along the course of the proliferating vessels.[11]

Figure 7: *FAF details an area of central hypofluorescence corresponding to the pigment plaque surrounded by a hyperfluorescent ring indicating tissue degradation.*



Speculation exists on whether the capillary abnormalities are the initial step in a process that leads to nutritional and metabolic damage to the retina, or if there is some sort of retinal change that precedes and leads to the formation of the abnormal capillaries. Müller cells garner the most suspicion due to their importance in maintaining proper health of the vasculature endothelium and sensory retina.[12,13] A primary dysfunction in these cells may lead to capillary abnormalities and retinal atrophy, with the superficial crystalline deposits representing remnants of degenerated Müller cells.[14]

SRNV formation appears to stem from proliferation and anastomoses that exist within the blood vessels of the retina. This differs from the retino-choroidal anastomoses found in macular degeneration. The reason for retinal capillary proliferation is unclear, but photoreceptor atrophy may give the capillaries easier access to the subretinal space.[15]

TREATMENT OPTIONS

Treatments for Type I IJT, or aneurysmal telangiectasia, aim at the destruction of the abnormal capillaries and aneurysms with photocoagulation or photodynamic therapy (PDT); or the stabilization of the blood retinal barrier with injectable medications. Limited options exist for those with Type II IJT due to the non-edematous nature of the disease, unless SRNV forms. Then treatments include laser photocoagulation, PDT, intravitreal triamcinolone injections, and intravitreal anti VEG-F agents.

Photocoagulation

Photocoagulation benefitted only those with Type I IJT, as noted by both Gass and Owakawa; and Gass and Blodi in their respective reports. They found a potential improvement in vision and exudation with photocoagulation. The procedure proved to be non-beneficial in those with Type II IJT.[3,16]

Photodynamic Therapy

Photodynamic therapy has also shown potential for improvement in vision, exudation, and edema in those with Type I IJT.[17]The benefits in Type II IJT, however, are limited to those with SRNV. Several reports show decreased leakage on FA as well as potential improvement or stabilization in vision in those with Type II with SRNV treated with PDT.[18,19,20,21] Even though there is no frank visual improvement in many of these cases, it is important to remember that non-treated SRNV has very poor visual prognosis with 80% having final visual acuities of worse than 20/200.[22] In those with Type II IJT without SRNV, treatment with PDT temporarily improved leakage on FA, but did not improve visual acuity.[23]

Intravitreal Injections

Injection of intravitreal triamcinolone decreases leakage in Type I IJT and Type II IJT both with and without SRNV. Some report improvement in both macular edema as shown with OCT and in vision in those with Types I and II IJT with SRNV. However, one must consider the high side effect profile with steroid injections. Additionally, even though leakage improves on FA in Type II IJT without SRNV, there is no improvement in retinal thickness with OCT or visual acuity in these cases. The effects of treatment in those who do show improvement are short lived with recurrent fluid leakage in three to six months.[24,25]

Similar to intravitreal triamcinolone, injection of anti VEG-F agents decrease FA leakage in Types I and II IJT with and without SRNV with a much more favorable side effect profile.[26,27,28,29,30,31] Reports indicate improvement in macular edema, retinal thickness, and vision in Types I and II IJT with SRNV. In Type II IJT patients without SRNV, few cases recorded improvement in retinal thickness and visual acuity; however, the majority failed to note a benefit in those with Type II IJT without SRNV. Although treatment with anti VEG-F has not been consistently shown to improve vision in those with Type II without SRNV, it is postulated that since it does reduce leakage from the abnormal blood vessels, that long-term therapy could potentially slow retinal atrophy. However, VEG-F is also needed in a certain level to maintain retinal vasculature health, and it is possible that long-term therapy could actually exacerbate retinal cell death.[32]

Due to the rare incidences of these conditions, most treatment studies available include a minimal number of patients or are even single case reports. Long-term follow up data is limited as well. Since there is such a poor untreated prognosis in those with SRNV in Type II, it is important to remember that stabilization of vision even without improvement could be a success in treatment. Additionally, substances that could slow the neurodegenerative process that appears to be taking place in Type II IJT are currently being investigated. One particular molecule, called ciliary neurotrophic growth factor, slows photoreceptor cell death in animal models and recently demonstrated safety in phase 1 clinical trials in humans.[33]At this time, however, treatment is reserved for those with SRNV in Type II IJT.

DIFFERENTIAL DIAGNOSES

Differential diagnosis for IJT should include other identifiable causes of macular telangiectasia such as diabetic retinopathy, retinal vein occlusion, carotid occlusive disease, radiation retinopathy, and others. Careful case history of potential systemic etiologies of retinal telangiectasia should be thoroughly considered. Diabetic retinopathy is likely the most commonly misattributed diagnosis for those with Type I IJT; however, the presence of fairly isolated, mostly temporal, unilateral retinal telangiectasia or hemorrhaging should raise a red flag for the diagnosis of Type I IJT.

Type II IJT is less commonly confused with other causes of retinal telangiectasia as it most often presents with one or more of its classic findings of crystalline deposits, retinal pigment plaques, right angle veins, and outer retinal atrophy. Due to the profound funduscopic appearance of the pigmented plaques and significant outer retinal atrophy on OCT, this condition can be confused with conditions such as age related macular degeneration (AMD), chronic or recurrent central serous retinopathy, toxic retinopathies from medications such as hydroxychloroquine, or retinal scarring from conditions such as presumed ocular histoplasmosis (POHS). Differentials in the formation of SRNV should include other conditions with a similar finding, such as wet AMD, POHS, polypoidal choroidal vasculopathy, and others.

VISUAL PROGNOSIS

Visual prognosis for IJT varies case to case. As discussed previously, vision loss in Type I IJT is related, at least initially, to the level of macular edema present, which can potentially show improvement with various treatment

modalities. However, due to the rarity of the condition, little information exists to determine long-term prognosis. The condition is a chronic one, and does require continued monitoring and possibly long-term treatment.

Vision loss from Type II IJT develops due to atrophy of the outer retina and photoreceptors. Due to the location of retinal atrophy being temporal to the foveal center, visual acuity often remains good in these patients until the development of SRNV. Data from the MacTel Study group showed that 42% of all patients had visual acuity of 20/25 or better.[34] However, this photoreceptor damage can create paracentral scotomas that lead to patient symptoms even with good retained visual acuity.[35] Various reports demonstrate that even with acceptable visual acuity, patients become symptomatic for patient perceived metamorphopsia, decreased reading speed, and difficulty in reading – the latter of which being the most frequently reported initial symptom.[36,37] These symptoms arise most commonly between the ages of 50 and 69. Exact values for long-term prognosis for Type II IJT varies between reports but patients do have increased risk for both decreased visual acuity as well as decreased quality of life from vision loss.[38,39]

CONCLUSION

Diagnosis of IJT can be complicated by clinical appearances mimicking other conditions. For example Type I IJT can easily be confused with diabetic retinal changes. While treatment options for the complication of macular edema from Type I IJT are essentially the same as those for diabetic macular edema, it is still important to make the correct diagnosis. While systemic conditions should still be considered and ruled out, a patient with Type I IJT could have unwarranted psychological distress if told they have retinal complications from diabetes when they don't have the disease.

Also, even though there is currently no known beneficial treatment in the case of Type II IJT without SRNV, it is still important to make an accurate assessment in order to educate patients properly about their condition and prognosis. Patients should be made aware that the condition is a progressive retinal disease which can lead to central vision loss, metamorphopsia, and paracentral scotomas. They also need to be thoroughly educated on how to properly monitor their vision monocularly, and to be examined periodically for SRNV. With proper understanding of IJT, optometrists can monitor these conditions appropriately, referring for additional treatment when necessary. ●

Table: Comparison of the Two IJT Classification Schemes

	Type I IJT		Type II IJT		Type III IJT	
	Gass and Blodi	Yanuzzi et al.	Gass and Blodi	Yanuzzi et al.	Gass and Blodi	Yanuzzi et al.
Subtypes	IA and IB	None	IIA and IIB	None	IIIA and IIIB	N/A
Gender prevalence	Male	Male	IIA – None IIB – Brothers	None	IIIA – female IIIB – male	N/A
Laterality	Unilateral	Unilateral	Bilateral	Bilateral	Bilateral	N/A
Stages	No	No	IIA – yes (five)	Yes (two)	No	N/A
Clinical findings	Both – easily visible retinal telangiectasia, exudation, macular edema IA > two clock hours of involvement IB < two clock hours of involvement	Easily visible retinal telangiectasia, exudation, macular edema	Both – minimally visible to no observable telangectiasia IA – Right angle veins, pigmented retinal plaques, superficial crystalline deposits, SRNV	Mild loss of retinal transperancy without obvious telangiectasia, pigment plaques, crystalline deposits, right angle vessels, SRNV	Occlusive capillary dropout, minimal exudation and edema IIIB – associated CNS disorders	N/A

References

1. Gass, J. D., and R. T. Owakawa. "Idiopathic Juxtafoveolar Retinal Telangiectasis." *Archives of Ophthalmology* 100.5 (1982): 769-80.
2. Issa, Peter Charbel, Mark C. Gillies, Emily Y. Chew, Alan C. Bird, Tjebo F. C. Heeren, Tunde Peto, Frank G. Holz, and Hendrik P. N. Scholl. "Macular Telangiectasia Type 2." *Prog Retin Eye Res* 34 (2013): 49-77.
3. Gass, J. Donald M., and Barbara A. Blodi. "Idiopathic Juxtafoveolar Retinal Telangiectasis." *Ophthalmology* 100.10 (1993): 1536-546.
4. Yannuzzi, Lawrence A. "Idiopathic Macular Telangiectasia." *Archives of Ophthalmology Arch Ophthalmol* 124.4 (2006): 450. Web.
5. Cohen, Steven M., Mark L. Cohen, Fayssal El-Jabali, and Scott E. Pautler. "Optical Coherence Tomography Findings In Nonproliferative Group 2A Idiopathic Juxtafoveal Retinal Telangiectasis." *Retina* 27.1 (2007): 59-66.
6. Sanchez JG, Garcia RA, Wu L, Berrocal MH, Graue-Wiechers F, Rodriguez FJ, et al. "Optical coherence tomography characteristics of group 2A idiopathic parafoveal telangiectasis." *Retina* 27 (2007): 1214–20.
7. Helb, Hans-Martin, Peter Charbel Issa, Rob L. P. Van Der Veen, Tos T. J. M. Berendschot, Hendrik P. N. Scholl, and Frank G. Holz. "Abnormal Macular Pigment Distribution In Type 2 Idiopathic Macular Telangiectasia." *Retina* 28.6 (2008): 808-16.
8. Bottoni F, Eandi CM, Pedenovi S, Staurenghi G. "Integrated clinical evaluation of Type 2A idiopathic juxtafoveolar retinal telangiectasis." *Retina* 30 (2010): 317–26.
9. Wong WT, Forooghian F, Majumdar Z, Bonner RF, Cunningham D, Chew EY. "Fundus autofluorescence in type 2 idiopathic macular telangiectasia: Correlation with optical coherence tomography and microperimetry." *Am J Ophthalmol* 148 (2009): 573–83.
10. Green, W. Richard, H. A. Quigley, Z. De La Cruz, and Bruce Cohen. "Parafoveal Retinal Telangiectasis." *Retina* 25.Supplement (2005): 162-70.
11. Eliassi-Rad, Babak, and W. Richard Green. "Histopathologic Study Of Presumed Parafoveal Telangiectasis." *Retina* 19.4 (1999): 332.
12. Powner, Michael B., Mark C. Gillies, Marina Tretiach, Andrew Scott, Robyn H. Guymer, Gregory S. Hageman, and Marcus Fruttiger. "Perifoveal Muller Cell Depletion in a Case of Macular Telangiectasia Type 2." *Ophthalmology* 117.12 (2010): 2407-416.
13. Gass, JD. "Müller cell cone, an overlooked part of the anatomy of the fovea centralis: Hypotheses concerning its role in the pathogenesis of macular hole and foveomacualr retinoschisis." *Arch Ophthalmol* 117 (1999) 821–823.
14. Wu, Lihteh. "Multimodality Imaging in Macular Telangiectasia 2: A Clue to Its Pathogenesis." *Indian J Ophthalmol Indian Journal of Ophthalmology* 63.5 (2015): 394.
15. Gass JD. "Chorioretinal anastomosis probably occurs infrequently in type 2A idiopathic juxtafoveolar retinal telangiectasis." *Arch Ophthalmol* 121 (2003): 1345–6.
16. Park, Donald W., Howard Schatz, H. Richard Mcdonald, and Robert N. Johnson. "Grid Laser Photocoagulation for Macular Edema in Bilateral Juxtafoveal Telangiectasis." *Ophthalmology* 104.11 (1997): 1838-846.
17. Kotoula, Maria G., Dimitrios Z. Chatzoulis, Constantinos H. Karabatsas, Aristoteles Tsiloulis, and Evangelia E. Tsironi. "Resolution of Macular Edema in Idiopathic Juxtafoveal Telangiectasis Using PDT." *Ophthalmic Surg Lasers Imaging Ophthalmic Surgery, Lasers, and Imaging* 40.1 (2009): 65-67.
18. Snyers, Bernadette, Claire Verougstraete, Laurence Postelmans, Anita Leys, and Philip Hykin. "Photodynamic Therapy of Subfoveal Neovascular Membrane in Type 2A Idiopathic Juxtafoveolar Retinal Telangiectasis." *American Journal of Ophthalmology* 137.5 (2004): 812-19.
19. Smithen, Lindsay M., and Richard F. Spaide. "Photodynamic Therapy and Intravitreal Triamcinolone for a Subretinal Neovascularization in Bilateral Idiopathic Juxtafoveal Telangiectasis." *American Journal of Ophthalmology* 138.5 (2004): 884-85.
20. Lee, Bailey L. "Bilateral Subretinal Neovascular Membrane In Idiopathic Juxtafoveolar Telangiectasis." *Retina* 16.4 (1996): 344-45.
21. Potter, Michael J., Shelagh M. Szabo, Eleanor Y. Chan, and Andrew H.c Morris. "Photodynamic Therapy of a Subretinal Neovascular Membrane in Type 2A Idiopathic Juxtafoveolar Retinal Telangiectasis." *American Journal of Ophthalmology* 133.1 (2002): 149-51.
22. Engelbrecht, Nicholas E. "Neovascular Membranes Associated With Idiopathic Juxtafoveolar Telangiectasis." *Archives of Ophthalmology Arch Ophthalmol* 120.3 (2002): 320.
23. Lahitte, Ghislaine Ducos De, Salomon Y. Cohen, and Alain Gaudric. "Lack of Apparent Short-term Benefit of Photodynamic Therapy in Bilateral, Acquired, Parafoveal Telangiectasis without Subretinal Neovascularization." *American Journal of Ophthalmology* 138.5 (2004): 892-94.
24. Alldredge, Claron D., and Bruce R. Garretson. "Intravitreal Triamcinolone For The Treatment Of Idiopathic Juxtafoveal Telangiectasis." *Retina* 23.1 (2003): 113-16.
25. Wu, Lihteh, Teodoro Evans, J. Fernando Arévalo, María H. Berrocal, Francisco J. Rodríguez, María Hsu, and Juan G. Sánchez. "Long-Term Effect Of Intravitreal Triamcinolone In The Nonproliferative Stage Of Type Ii Idiopathic Parafoveal Telangiectasia." *Retina* 28.2 (2008): 314-19.
26. Kovach, Jaclyn L., and Philip J. Rosenfeld. "Bevacizumab (Avastin) Therapy For Idiopathic Macular Telangiectasia Type Ii." *Retina* 29.1 (2009): 27-32.
27. Júnior, Otacílio Oliveira Maia, Maria Teresa Brizzi Chizzotti Bonanomi, Walter Yukihiko Takahashi, Vinícius Paganini Nascimento, and Beatriz Sayuri Takahashi. "Intravitreal Bevacizumab for Foveal Detachment in Idiopathic Perifoveal Telangiectasia." *American Journal of Ophthalmology* 144.2 (2007): 296-99.
28. Issa, P. C., R. P. Finger, F. G. Holz, and H. P N Scholl. "Eighteen-month Follow-up of Intravitreal Bevacizumab in Type 2 Idiopathic Macular Telangiectasia." *British Journal of Ophthalmology* 92.7 (2008): 941-45.
29. Gamulescu, Maria-Andreea, Andreas Walter, Helmut Sachs, and Horst Helbig. "Bevacizumab in the Treatment of Idiopathic Macular Telangiectasia." *Graefes Arch Clin Exp Ophthalmol Graefe's Archive for Clinical and Experimental Ophthalmology* 246.8 (2008): 1189-193.
30. García-Ben, A., F. Gómez-Ulla, and M.j. Rodriguez-Cid. "Intravitreal Bevacizumab in the Treatment of Idiopathic Juxtafoveal Telangiectasis Type I." *Archivos De La Sociedad Española De Oftalmología (English Edition)* 89.7 (2014): 269-71.
31. Nakhwa, Chinmayp, and Manavid Sindal. "Idiopathic Macular Telangiectasia Type 1 with Ruptured Retinal Arterial Macroaneurysm Post Intravitreal Bevacizumab." *Middle East African Journal of Ophthalmology Middle East Afr J Ophthalmol* 22.3 (2015): 396.

32.) Kupitz, Elke H., Tjebo F. C. Heeren, Frank G. Holz, and Peter Charbel Issa. "Poor Long-Term Outcome Of Anti-Vascular Endothelial Growth Factor Therapy In Nonproliferative Macular Telangiectasia Type 2." *Retina* 35.12 (2015): 2619-626.

33. Chew, Emily Y., Traci E. Clemons, Tunde Peto, Ferenc B. Sallo, Avner Ingerman, Weng Tao, Lawrence Singerman, Steven D. Schwartz, Neal S. Peachey, and Alan C. Bird. "Ciliary Neurotrophic Factor for Macular Telangiectasia Type 2: Results From a Phase 1 Safety Trial." *American Journal of Ophthalmology* 159.4 (2015): 659-666.
34. Clemons, Traci E., Marc C. Gillies, Emily Y. Chew, Alan C. Bird, Tunde Peto, Maria J. Figueroa, and Molly W. Harrington. "Baseline Characteristics of Participants in the Natural History Study of Macular Telangiectasia (MacTel) MacTel Project Report No. 2." *Ophthalmic Epidemiology* 17.1 (2010): 66-73.
35. Heeren, Tjebo F. C., Traci Clemons, Hendrik P. N. Scholl, Alan C. Bird, Frank G. Holz, and Peter Charbel Issa. "Progression of Vision Loss in Macular Telangiectasia Type 2." *Investigative Opthalmology & Visual Science Invest. Ophthalmol. Vis. Sci.* 56.6 (2015): 3905.
36. Heeren, Tjebo F. C., Frank G. Holz, and Peter Charbel Issa. "First Symptoms And Their Age Of Onset In Macular Telangiectasia Type 2." *Retina* 34.5 (2014): 916-19.
37. Finger, Robert P., Peter Charbel Issa, Rolf Fimmers, Frank G. Holz, Gary S. Rubin, and Hendrik P. N. Scholl. "Reading Performance Is Reduced by Parafoveal Scotomas in Patients with Macular Telangiectasia Type 2." *Investigative Opthalmology & Visual Science Invest. Ophthalmol. Vis. Sci.* 50.3 (2009): 1366.
38. Watzke, Robert C., Michael L. Klein, James C. Folk, Samuel G. Farmer, Richard S. Munsen, Robert J. Champfer, and K. R. Sletten. "Long-Term Juxtafoveal Retinal Telangiectasia." *Retina* 25.6 (2005): 727-35.
39. Lamoureux, Ecosse L., Rebecca M. Maxwell, Manjula Marella, Mohamed Dirani, Eva Fenwick, and Robyn H. Guymer. "The Longitudinal Impact of Macular Telangiectasia (MacTel) Type 2 on Vision-Related Quality of Life." *Investigative Opthalmology & Visual Science Invest. Ophthalmol. Vis. Sci.* 52.5 (2011): 2520.

Innovations in Eye Care: Sport Vision

Dr. Graham Erickson *has been on the faculty of Pacific University since 1998, and currently teaches the Pediatric Optometry, Vision Therapy and Sports Vision courses. He has authored the textSports Vision: Vision Care for the Enhancement of Sports Performance, as well as co-authoring the text OptometricManagement of Reading Dysfunction, and published chapters and articles in various optometric journals. He lectures nationally and internationally on the topics of Sports Vision, pediatrics, binocularvision, and dyslexia.*

Optometry has a long tradition of providing vision care services to optimize daily function. While this has often focused on academic and occupational performance, sports performance is another important area where vision plays a critical role. Athletes, trainers and coaches have recognized that excellent vision is an important aspect of performance, and more athletes and teams are looking for ways to optimize visual performance. While an interest in sports vision is not new, there have been some recent innovations in instrumentation that utilize new digital technology.

VISUAL PERFORMANCE ASSESSMENT

Many studies have found that higher-achieving athletes perform better on many measures of visual performance than non-athletes or lower-achieving athletes. Recently, several companies have developed instruments that measure various aspects of visual performance, and such performance can be compared to a database of performance by other athletes. Computerized assessment and training devices such as the Senaptec Sensory Station (http://senaptec.com), Sports Vision Performance from M&S® (http://www.mstech-eyes.com/products/category/sports-vision-performance), RightEye (https://www.righteye.com/tests-therapies/vision-performance) and Vizual Edge Performance Trainer® (http://vizualedge.com) have been developed to measure a broad range of visual, cognitive and sensorimotor skills.

The Senaptec Sensory Station is a successor to the Sensory Station device originally developed by Nike Inc. Research with the Nike version of this instrument has demonstrated that certain assessments in the battery are reliable and cross-validated measures that can be used to investigate sensorimotor abilities in relation to performance in sports.[1,2] Furthermore, worse performance on the Sensory Station has been associated with an increased likelihood of sustaining head impacts during practices and games among US collegiate football players,[3] indicating a link between collision avoidance and visual-motor skills. This suggests that these assessments might be useful for proactively assessing the risk of concussion, as well as potentially measuring the recovery of visual performance following a concussive episode. While there is limited evidence regarding the reliability or validity of the other systems, many of these systems employ standard psychophysical protocols, and thus it is reasonable to expect that the measurements are reliable.

SPORTS VISION TRAINING

Sports vision training (SVT) programs operate under the logic that practice with demanding visual, perceptual and sensorimotor tasks will improve vision, leading to quicker sensory processing, swifter and more accurate motor movements, and improved athletic performance while also potentially reducing injury. SVT approaches have been advanced greatly by training programs that use information about the structure and function of the visual system combined with recent innovations in perceptual learning paradigms to engender more specific and robust learning. Virtual reality (VR) simulations that can recreate and augment sporting contexts to promote certain sports-specific visual-cognitive abilities have also enhanced SVT approaches.

A recent innovation in visual component training is called Ultimeyes® (https://ultimeyesvision.com). This video application incorporates diverse stimuli, adaptive near-threshold training with learning-optimized flickering stimuli, and multisensory feedback in a digital training program designed to improve foundational aspects of visual sensitivity. In a series of studies, this training app has been shown to improve visual acuity and contrast sensitivity in both non-athletes[4] and athletes,[5] as well as to improve batting[5] and pitching[6] performance in collegiate baseball players.

The CogniSense NeuroTracker (https://neurotracker.net) is an example of a perceptual-cognitive training program. The training platform entails an immersive three-dimensional "multiple object tracking" program to increase cognitive load. There has been ample research with the NeuroTracker system in groups of healthy young adults,[7] healthy older adults,[8,9] and athletes across several sports and skill levels. NeuroTracker performance has been correlated with actual game performance in professional basketball players,[10] and training with this program has been demonstrated to selectively transfer to improved small-sided game performance in university-level soccer players.[11]

Actual sports practice is typically viewed as the most natural method for developing the necessary skills for success. However, practice sessions have the potential for injury to the athlete. Over the past several years, computerized simulations and VR platforms have been developed to simulate game action, and are now considered a type of natural sports training. Such simulation platforms allow for the design of complex training protocols that can mimic real-game activities, allowing athletes to gain 'mental repetitions.' Three companies in particular, Eon Sports VR (http://eonsportsvr.com/), StriVR Labs (http://www.strivrlabs.com), and Axon Sports (http://www.axonsports.com/), have recently developed suites of digital training simulations that are marketed towards athletes, coaches and trainers. In addition to these broad commercial platforms that have applications for many different sports, there is a growing number of products that target specific individual sports. It is important to note that these VR sport simulations are a new technology with relatively little supporting evidence at this time.

SUMMARY

This brief summary has highlighted some of the recent innovations in sports vision. More detailed information can be found in a recently published review paper.[12] These innovations provide options to help your athletic patients see their sport more clearly. ●

References

1. Erickson GB, Citek K, Cove M, et al. Reliability of a computer-based system for measuring visual performance skills. Optometry 2011, 82(9):528-42.
2. Wang L, Krasich K, Bel-Bahar T, Hughes L, Mitroff SR, Appelbaum LG. Mapping the structure of perceptual and visual-motor abilities in healthy young adults. Acta Psychol (Amst) 2015; 157:74-84.
3. Harpham JA, Mihalik JP, Littleton AC, Frank BS, Guskiewicz KM. The effect of visual and sensory performance on head impact biomechanics in college football players. Ann Biomed Eng 2014, 42:1-10.
4. Deveau J, Lovcik G, Seitz AR. Broad-based visual benefits from training with an integrated perceptual-learning video game. Vision Res 2014; 99:134-40.
5. Deveau J, Ozer DJ, Seitz AR. Improved vision and on-field performance in baseball through perceptual learning. Curr Biol 2014; 24(4):R146-7.
6. Deveau J, Thurman S, Seitz AR. Improvements in baseball pitching through vision training. *In press*
7. Parsons B, Magill T, Boucher A, et al. Enhancing cognitive function using perceptual-cognitive training. Clinical EEG and Neuroscience 2016; 47:37-47.
8. Legault I, Allard R, Faubert J. Healthy older observers show equivalent perceptual-cognitive training benefits to young adults for multiple object tracking. Front Psychol 2013; 4:323.
9. Legault I, Faubert J. Perceptual-cognitive training improves biological motion perception: Evidence for transferability of training in healthy aging. Neuroreport 2012; 23:469-73.
10. Mangine GT, Hoffman JR, Wells AJ, et al. Visual tracking speed is related to basketball-specific measures of performance in NBA players. J Strength Conditioning Res 2014; 28:2406-14.
11. Romeas T, Faubert J. Soccer athletes are superior to non-athletes at perceiving soccer specific and non-sport specific human biological motion. Front Psychol 2015; 6:705.
12. Appelbaum LG, Erickson G. Sports vision training: A review of the state-of-the-art in digital training techniques. Int Rev Sport Exercise Psychol 2016; DOI: 10.1080/1750984X.2016.1266376

Every Member of Your Team is a Salesperson

Pauline Blachford *consults with optometrists across North America on how to reduce unbooked appointments, increase eyewear sales, and improve employee engagement and productivity. She coaches optometric staff on strategies and techniques for effectively recalling patients, and has developed a workshop designed to empower employees with the tools to contribute valuably to their clinic. Pauline writes regularly about practice management for the Canadian Journal of Optometry and is frequently invited to speak at industry conferences and events across the continent. For more information or to connect, visit www.paulineblachford.com.*

In my two decades working on the business side of this industry, I've seen how it can be a challenge for small, independent optometry practices to employ a full-time optician to oversee eyewear sales. Even at larger practices where there may be several opticians, it can be a struggle to significantly increase sales without a proper strategy in place. Whether a practice's budget, staff and resources are small or great, the most cost-effective, productive and successful way to boost eyewear sales is to convert your entire optometry team into a team of capable, confident salespeople.

At any practice, the OD, optician, recallers and front-desk staff have the eye health, eye care or patient expertise to effectively ensure a patient's eye care needs are met. That knowledge can also be applied to make eyewear sales a team initiative that leverages staff insight and utilizes existing resources, all while prioritizing patient needs and boosting the practice's bottom line.

There is a learning curve inherent to implementing a strong sales strategy in any industry: finding the time to sell, personalizing sales pitches, communicating value, and ensuring efficiency are challenges that will need to be overcome.[1] However, the prospect of increased eyewear sales and greater revenue should be worth the time, patience and effort. Here are three ways to begin turning your existing optometry team into an effective and profitable team of sales staff.

ENSURE EVERYONE AT YOUR PRACTICE KNOWS YOUR EYEWEAR PRODUCTS

Whether it's types of frames, prices, colours or warranty, the more information your staff members have about the products you sell, the more likely they will be able to sell them.[2]

Your recallers or front-desk staff do not need to become experts, but they should have access to product information – and be given time to learn it – so that they can answer patient questions, and identify, respond to and note patient eyewear needs while booking appointments or chatting with patients in the waiting room. Having more informed employees also means that during vacations, illness or turnover, your practice's eyewear sales strategy doesn't get put on hold.

Encourage employees to spend time familiarizing themselves with your practice's products. Bringing in sales representatives to speak to your full team, reviewing testimonials, reading market literature and physically looking at and using some of the products you have in stock are good ways to get started on expanding your team's knowledge.[3]

GET YOUR TEAM LEVERAGING EACH AND EVERY OPPORTUNITY

Every time a patient calls, emails or enters your practice, it's an opportunity to have a positive impact that could lead to a sale.[4] While booking an appointment over the phone, a recaller should feel empowered to ask a patient about their current eyewear, their eyewear needs and contact lens refills, as appropriate. He or she should also remind patients to bring in their existing glasses.

Selling a product like eyewear is easier with visuals. Eyewear should be attractively displayed in an area – such as the waiting room – that makes it easy for patients to browse at their leisure. You can also have your staff wear products that are sold at your practice – a promotional opportunity that can easily be included as an employee benefit.[5]

Finally, an OD should discuss eyewear with the patient during their eye health exam, and personally introduce them to the practice's optician for further questions. This helps transfer patient-doctor trust to other members of the optometry team.

LISTEN ACTIVELY, TAKE NOTES AND SHARE YOUR FEEDBACK WITH THE TEAM

Thinking like a strategic salesperson means thinking about how you can better help and serve your customers. To do so effectively means to first collect as much information as possible: Are your patients on a budget? Do they have insurance plans that include funds for eyewear? Have they expressed concerns about their current products?

Have your staff ask open-ended questions, and engage patients in conversations about their needs, concerns and thoughts.[6] Any time new information is learned, have a process for adding that to a patient's file in such a way that it can be accessed by all team members. Another way to do this is to have a recaller let the OD and optician know that a patient will be bringing in their eyewear, and are interested in looking at new products. This ensures the entire team is prepared to discuss eyewear options as soon as the patient visits.

NEXT STEPS

Investment in staff is one of the most important factors of all when it comes to ensuring the success of your budding sales team.[7] Hiring a sales professional to train your staff on how to communicate value, actively listen to patient needs, and close a sale could help get your team's skill level to the next level.

Keep sales top-of-mind in meetings, and encourage regular conversations about products, sales and experiences. You may have a staff member with past experience in retail who can share their knowledge with their colleagues. Regular check-ins with your team also provide a forum where questions, insights and experiences can be shared and discussed, to the benefit of the entire team.

Finally, setting a clear goal for increasing your eyewear sales and tracking your progress is a critical. Only 28% of organizations that prepare progress reports actually use the data they collect to improve their training for sales representatives.[8] Sharing eyewear sales information and patient feedback, and using it to improve your strategy, will empower your team to effectively achieve your eyewear sales goals, while giving your practice a competitive edge when it comes to sales. ●

REFERENCES

1 Faust C. "The Three Biggest Challenges of Today's Sales Reps", MarketingProfs, May 26, 2015. Retrieved from http://www.marketingprofs.com/opinions/2015/27725/the-three-biggest-challenges-of-todays-sales-reps

2 Connick W. "How to Sell Any Product", The Balance, September 30, 2016. Retrieved from https://www.thebalance.com/how-to-sell-any-product-2917619

3 Hudson M. "The Benefits of Product Knowledge", The Balance, February 1, 2017. Retrieved from https://www.thebalance.com/benefits-of-product-knowledge-2890302

4 Downes M. "Everybody's a salesperson", Financial Times, October 5, 2012. Retrieved from: https://www.ft.com/content/6aeb63c0-e535-11e1-8ac0-00144feab49a

5 "Five Points of Selling High-End Eyewear by Optylux", The Optical Vision Site, June 24, 2009. Retrieved from: http://www.theopticalvisionsite.com/sales/five-points-of-selling-high-end-eyewear-by-optylux/

6 Parinello T. "How to Sell in 60 Seconds", Entrepreneur. Retrieved from: https://www.entrepreneur.com/article/65972

7 Downes M. "Everybody's a salesperson", Financial Times, October 5, 2012. Retrieved from: https://www.ft.com/content/6aeb63c0-e535-11e1-8ac0-00144feab49a

8 Faust C. "The Three Biggest Challenges of Today's Sales Reps", MarketingProfs, May 26, 2015. Retrieved from http://www.marketingprofs.com/opinions/2015/27725/the-three-biggest-challenges-of-todays-sales-reps

Shire

From **innovation** to **inspiration**.

Shire is bringing its expertise to ophthalmics.

Keep your eyes on Shire.

PRMCDA/CA//0028 March 2017

FEEL LIKE YOU'RE WEARING NOTHING AT ALL.

The first and only Water Gradient Contact Lens. visit **feels**like**nothing**.ca to learn more.

Alcon A Novartis Division