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Case Report

Visual Therapy in Traumatic Third Nerve Palsy as an Interdisciplinary Intervention: A Mexican Case Report

Terapia visual y rehabilitación multidisciplinaria de la parálisis del tercer nervio craneal: reporte de caso mexicano

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ABSTRACT

Introduction: Traumatic brain injury (TBI) frequently affects the third cranial nerve, causing oculomotor alterations such as limited adduction, ptosis, and mydriasis. Motor, sensory, and perceptual deficits in these patients are rarely rehabilitated, as treatment usually focuses on surgery and anti-inflammatory therapies. After an interdisciplinary management approach, this article describes the clinical evolution of sensory, motor, and perceptual skills of a 26-year-old male with a third nerve palsy secondary to a TBI. *Case Report*: A 26-year-old male presented with left-eye 60Δ exotropia, diplopia, balance issues, photophobia, superior visual field defects, and headaches caused by a TBI sustained four months earlier. A personalized optometric visual therapy program was designed and implemented to rehabilitate the patient's oculomotor, sensory, and perceptual visual skills. The interdisciplinary approach included Botox therapy administered by strabismologists. *Conclusions*: The combination of optometric visual therapy and Botox effectively improved the patient's symptoms, restoring binocular, sensory, and perceptual visual skills to near-normal levels. These results remained stable months after treatment. Further physiological studies are needed to clarify the mechanism of action of optometric visual therapy, as it could reduce or eliminate the need for repeated Botox applications in patients with third nerve palsy caused by brain trauma. Interdisciplinary approaches are crucial for maximizing therapeutic benefits and enhancing patients' quality of life.

Keywords: third nerve palsy; strabismus; traumatic brain injury; visual therapy

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RESUMEN

Introducción: Las lesiones cerebrales traumáticas (LCT) afectan con frecuencia al tercer par craneal, causando alteraciones oculomotoras como aducción limitada, ptosis y midriasis. Los déficits motores, sensoriales y perceptivos en estos pacientes rara vez se rehabilitan, ya que el tratamiento suele centrarse en la cirugía y las terapias antiinflamatorias. Tras un enfoque de tratamiento interdisciplinario, este artículo describe la evolución clínica de las habilidades sensoriales, motoras y perceptivas de un varón de 26 años con parálisis del tercer nervio secundaria a una LCT. *Informe del caso*: Un varón de 26 años presentaba exotropia de 60Δ en el ojo izquierdo, diplopía, problemas de equilibrio, fotofobia, defectos del campo visual superior y dolores de cabeza causados por un traumatismo craneoencefálico sufrido cuatro meses antes. Se diseñó y aplicó un programa personalizado de terapia visual optométrica para rehabilitar las habilidades visuales oculomotoras, sensoriales y perceptivas del paciente. El enfoque interdisciplinario incluyó la terapia con Botox administrada por estrabólogos. *Conclusiones:* La combinación de terapia visual optométrica y Botox mejoró eficazmente los síntomas del paciente, restaurando las habilidades visuales binoculares, sensoriales y perceptivas a niveles casi normales. Estos resultados se mantuvieron estables meses después del tratamiento. Se necesitan más estudios fisiológicos para aclarar el mecanismo de acción de la terapia visual optométrica, ya que podría reducir o eliminar la necesidad de aplicaciones repetidas de Botox en pacientes con parálisis del tercer nervio causada por un traumatismo cerebral. Los enfoques interdisciplinarios son cruciales para maximizar los beneficios terapéuticos y mejorar la calidad de vida de los pacientes.

Palabras clave: parálisis del tercer nervio; estrabismo; lesión cerebral traumática; terapia visual.

INTRODUCTION

The third cranial nerve originates from the common oculomotor nucleus located in the mesencephalon and passes through the superior orbital fissure. It innervates the levator palpebrae muscle and the extraocular muscles (superior rectus, inferior rectus, medial rectus, and inferior oblique). Additionally, through its parasympathetic component, it facilitates miosis and accommodation (1).

Third nerve palsy (TNP) can be classified based on several criteria, including extent, etiology, pupillary involvement, and lesion location. In terms of extent, it can be complete, affecting all innervated muscles, or incomplete, involving only some. Based on etiology, it may be congenital, due to developmental anomalies, or acquired, caused by trauma, vascular diseases, infections, tumors, or aneurysms. Pupillary involvement distinguishes between pupil-involving palsy, often from compressive lesions (e.g., aneurysms), and pupil-sparing palsy, usually from microvascular causes (e.g., diabetes) (2). According to location, it may be nuclear (midbrain damage), fascicular (brainstem fibers), or peripheral (extracranial path) (2,3). Accurate classification is crucial for effective diagnosis, management, and prognosis.

TNP secondary to traumatic brain injury (TBI) has an incidence of 2.7 % (4). It is characterized by limited adduction, ptosis, and mydriasis, often resulting in ocular complications, such as accommodative dysfunction (42.8 %) and convergence insufficiency (36.3 %) (5). These conditions can significantly reduce the patient's quality of life due to symptoms such as diplopia, asthenopia, and deficits in depth perception (5,6).

The management of TNP begins with medical and surgical interventions, such as the application of botulinum toxin within 2 to 3 weeks after onset (7). This approach has been shown to be effective in restoring ocular alignment and fusion in 76.7 % of patients, although it demonstrates poor outcomes in stereopsis recovery (240 arcseconds) (8). Optometric examination of TBI should begin with measuring visual acuity, followed by assessing pupillary reflexes, refraction, binocular and oculomotor status, accommodative function, color vision, visual fields, and ocular health (9).

Regarding the management of TNP ocular sequelae, some authors recommend spectacle prescriptions (considering the use of tints and prisms), optometric vision therapy for fixation, accommodative, and oculomotor dysfunctions (including vergence, saccades, and pursuits), and rehabilitation of visual fields (to enhance awareness of the residual visual field, specifically hemianopsias and quadrantanopsias, if present) (9,10).

Optometric vision therapy, incorporating binasal occlusion, base-in prisms for convergence insufficiency, yoked prisms for egocentric neglect, and filters for photophobia, has demonstrated effectiveness in managing non-strabismic accommodative and vergence disorders (11). However, the evidence supporting its efficacy remains limited (12).

Currently, there is a lack of documented studies involving interdisciplinary interventions among ophthalmologists, optometrists, and other specialties aimed at rehabilitating patients with TNP secondary to TBI. Existing literature often describes collaborations between ophthalmologists and physical therapists (13); however, this approach may be insufficient for comprehensive visual rehabilitation, as it does not incorporate the specialized expertise and tools that optometrists provide. A truly multidisciplinary approach, involving ophthalmologists, optometrists, physical therapists, and other relevant specialists, is essential to address the complex visual and neurological sequelae of TNP following TBI.

This article reports the clinical evolution of sensory, motor, and perceptual skills in a 26-year-old male with TNP secondary to TBI following an interdisciplinary management approach.

CASE REPORT

In November 2019, a 26-year-old male presented with left-eye strabismus, diplopia, balance disorders, photophobia, superior visual field defects, and headache secondary to a TBI caused by a vehicle rollover accident. These symptoms had progressed over a four-month period.

The hospital discharge diagnosis included severe TBI, multiple brain contusions with diffuse axonal damage, left intraventricular hemorrhage, pneumonia, and complete palsy of the third cranial nerve. After the initial treatment, the patient was discharged and later admitted to a local rehabilitation and physiotherapy center.

A comprehensive optometric assessment was conducted (Table 1), and a vision therapy program was designed to address oculomotor, sensory, and perceptual visual symptoms. The program was based on Elizabeth Caloroso's Strabismus Management Protocol for optometric strabismus intervention (14) (Table 2).

TABLE 1. **Thirty-One Months of Follow-Up on Clinical Findings**

	Baseline	After 14 months (before Botox)	After 18 months	After 31 months	Post-VT
Worth Four Dot Test	Alternating suppression	Crossed diplopia	Suppression OS (D) Fusion (N)	Fusion (D & N)	Fusion (D & N)
Stereopsis	Null stereopsis	Null stereopsis	400 arcseconds	50 arcseconds	40 arcseconds
EOM evaluation	OS: Medial rectus muscle -3 Upward gaze restriction -2 Downward gaze restriction -1 Ptosis	OS: Upward and downward gaze restriction -1	OS: Upward and downward gaze restriction -1	OS: Upward and downward gaze restriction -1	OS: Upward and downward gaze restriction -1
PBCT	D: left hypertropia, >60Δ exotropia N: left hypertropia, >60Δ exotropia	D: 6Δ left hypertropia, 18Δ exotropia N: 9Δ left hypertropia, 7Δ exotropia	D: 4Δ left hypertropia, 6Δ exotropia N: 4Δ left hypertropia, 15Δ exophoria	D: 4Δ left hypertropia N: 1Δ left hypertropia, three exophoria	D: 1Δ left hypertropia N: 2Δ left hypertropia, four exophoria
Flippers accommodative facility (+2.00/–2.00 D)	Not measured	Not measured	OD: 2 cpm OS: 0 cpm OU: 3 cpm	OD: 17 cpm OS: 16 cpm OU: 18 cpm	OD: 19 cpm OS:17 cpm OU: 13 cpm

	Baseline	After 14 months (before Botox)	After 18 months	After 31 months	Post-VT
Amplitude of accommodation	Not measured	Not measured	OD: 14 D OS: 3.33 D	OD: 7.14 D OS: 6.25 D	OD: 9.52 D OS: 7.14 D
MEM	Not measured	Not measured	OD: -1.00 D OS: +1.00 D	OD: 0.00 D OS: +1.00 D	OD: +0.50 D OS: +1.25 D
NRA	Suppression ---not measured	Diplopia --- not measured	+1.75 D	+2.75 D	+2.25 D
PRA	Suppression ---not measured	Diplopia --- not measured	-1.00 D	-1.50 D	-4.75 D
NFV (Distance)	Suppression --- fusional ranges not measured	Diplopia --- fusional ranges not measured	Suppression --- fusional ranges not measured	2/3/2	5/6/3
NFV (Near)	Suppression --- fusional ranges not measured	Diplopia --- fusional ranges not measured	X/6/0	10/13/10	8/9/5
PFV (Distance)	Suppression --- fusional ranges not measured.	Diplopia --- fusional ranges not measured	Suppression --- fusional ranges not measured	3/4/2	2/3/2
PFV (Near)	Suppression --- fusional ranges not measured.	Diplopia --- fusional ranges not measured	X/9/3	3/5/3	12/14/10
AC/A ratio	Suppression --- fusional ranges not measured	Diplopia --- fusional ranges not measured	1/1 (Gradient Method)	4/1 (Gradient Method)	3/1 (Gradient Method)
DEM horizontal time (seconds)	Suppression --- fusional ranges not measured	Diplopia --- fusional ranges not measured	39 s	35 s	36 s
DEM vertical time	Suppression --- fusional ranges not measured	Diplopia --- fusional ranges not measured	47 s	37 s	37 s
Visual perceptual skills (tvps) percentile range	---	---	---	SR = 16 % SM = 25 %	SR = 50 % SM = 2 %

Note. Extraocular muscle (EOM), prism bar cover test (PBCT), diopters (D), monocular estimate method (MEM), negative relative accommodation (NRA), positive relative accommodation (PRA), negative fusional vergence (NFV), positive fusional vergence (PFV), accommodative convergence/accommodation ratio (AC/A ratio), developmental eye movement test (DEM), test of visual perception skills (TVPS), spatial relationships (SR), sequential memory (SM), visual therapy (VT).

Monocular intervention was performed during the first year following the trauma (Table 2). During this period, exotropia decreased to 18Δ at distance and 7Δ at near, with hypertropia of 6Δ at distance and 9Δ at near. Alternating suppression was eliminated, but diplopia persisted. A referral to a strabismus specialist was made for surgical treatment or botulinum toxin application. At this appointment, an orbital floor fracture with muscle entrapment was ruled out via orbital tomography, and the specialist prescribed a botulinum toxin injection of 5 units in both eyes, scheduled for January 27, 2021.

Subsequently, biocular and binocular visual rehabilitation was initiated (Table 2). A computerized visual field evaluation and the Visual Perceptual Skills Test were incorporated to detect visual field and spatial perception alterations related to the TBI. Perceptual intervention and maintenance of motor and sensory skills were also provided (see Table 2). After 18 months of therapy, the patient's sensory (fusion and stereopsis) and visuoperceptual functions improved progressively and significantly (Table 1). Notably, these improvements remained stable without the need for a second dose of Botox (Table 1).

TABLE 2. **Optometric Visual Therapy Techniques**

INTERVENTION	OFFICE-BASED THERAPY	HOME THERAPY
Monocular intervention (for 1 year): 1 hour per day Note: Left eye occlusion with translucent tape has been indicated for the rest of the day	• Accommodation flexibility exercises (Hart chart) • Eye tracking (Marsden ball, spotlight tracking) • Eyes, hands, and feet coordination (bouncing small rubber balls, picking up marbles with toes) • Saccadic eye movements (saccadic charts)	• Hart chart • Marsden ball • Bouncing small rubber balls • Picking up marbles with toes • Putting rice into a straw • Saccadic charts
Biocular (spanning 5 months) and binocular intervention (spanning 2 months): 1 hour per day twice a week	• Gross convergence (red-green mat "pigeon feet," red and green non-variable anaglyphs with auxiliary prisms) • Hand-eye coordination (bouncing and catching rubber balls, throwing balls into a basket) • Fine convergence (Brock string, Bernell nine positions of gaze chart (Bernell ®) plus prisms) • Oculomotor tracking (Van Order Star, Marsden ball) • Inciclotorsion and exciclotorsion (Torsion disparity trainer (Bernell ®)) • Accommodative facility (Hart Chart, flippers through jumps of +/- 0.50 to +/-2.00)	• Supra-convergence (punctual monitoring with star pictures on the roof, plus red-green filter, and Brock string) • Convergence at the primary position of gaze (Brock string) • Eye-hand coordination (throwing balls in boxes 15 minutes daily, yo-yo with both left and right hands) • Antisuppression therapy (Amblyo Match app (Sterea Ciprian)/Tetris app (Invagapp), reading with red-green grille)
Perceptual and maintenance of sensory and motor skills intervention: For 12 months	• Balance (balance board with yoked prisms in the lower field) • Tangent screen + visual stimulus with intermittent lights (touching the visual stimulus, calculating its location on the tangent screen in a monocular or binocular way) • Marsden ball + Hart chart (catching the ball while looking at the primary position of gaze with the Hart chart, calculating the location with peripheral vision) • Speed response + illuminated switches, peripheral vision (Touch the stimulus—Tap it FM ®), which activates using only peripheral vision for perception • Starting with a visual stimulus focused on improving spatial-location skills through the following battery: sunny visual motor series A, B, C, 1B	• Red and green non-variable anaglyphs with auxiliary prisms • Hand-eye coordination (bouncing and catching rubber balls, throwing balls into a basket) • Marsden ball • Torsion disparity trainer (Bernell ®) • Bosu ball plus heart chart

Note. Phases of visual rehabilitation and optometric techniques focused on recovering the patient's motor, sensory, and perceptual abilities

DISCUSSION

It has been claimed that in most cases of TBI, patients typically improve on their own over time. However, there is evidence of no "spontaneous improvement" following more than 1 month after the injury (15). Disregarding the rehabilitation of motor, sensorial, and perceptual skills could negatively impact the performance of daily activities and the patient's quality of life (16). Binocular vision is required to exit physical, vestibular, cognitive, speech, and occupational therapy, and should be closely monitored and rehabilitated.

Therefore, in this case, optometric visual therapy started three months after a TBI, contrary to the traditional recommendations, which speak of intervention after a year of trauma (17). We observed positive changes since the first stage of intervention, enabling the patient to recognize and reorganize the information received through fixations, saccades, and accommodation stimulation (Table 1).

Following the recommendations of (14), Botox was applied during the biocular stage to enhance fusion capacity, resulting in the successful restoration of the patient's stereopsis. A recent meta-analysis reports that Botox is effective, particularly in acute scenarios, while its efficacy may diminish in chronic conditions (7). However, it is essential to note that although the meta-analysis measures diplopia, the included studies generally do not assess stereopsis (a critical factor addressed in our study). In this case, the improvement in stereopsis, linked to enhanced fusion and relief of diplopia, confirms the value of combining Botox with optometric vision therapy.

Interestingly, including motor experience in optometric vision therapy (particularly in binocular and perceptual interventions) resulted in significant improvements in the flexibility and amplitude of accommodation, vergence, ocular movements, and perceptual skills (Table 1). These findings can likely be attributed to neuroplasticity mechanisms stimulated by motor behavioral experiences (18). Specifically, these improvements may be associated with the effects of acute aerobic exercise, such as shorter bouts of high-intensity interval training or longer bouts of lower-intensity continuous exercise. These activities enhance motor cortex excitability and promote subcortical neuroplasticity, particularly in the hippocampus, by increasing neurotrophic factors like the brain-derived neurotrophic factor (BDNF), ultimately leading to more effective synaptic connections (19).

There is currently evidence that voluntary exercise can endogenously upregulate many neurotrophic factors involved in neuronal survival (17,20). For example, daily exercise has been shown to prevent and protect against age-related brain damage by increasing insulin-like growth factor I (IGF-I) (21). Additionally, an in vitro study has demonstrated that the administration of neurotrophic factors, such as nerve growth factor (NGF) and ciliary neurotrophic factor (CNTF), can promote ocular muscle cell proliferation and reduce inflammatory levels, producing a protective effect on muscle cell function in paralytic strabismus (22). In the same way, voluntary exercise increases BDNF production in the dorsal (motor) striatum, through dopamine stimulation (23) and administration of exogenous BDNF in mice can retard motor dysfunction in motor neuron disease and diminish denervation muscle atrophy and motor axon loss (24) and can even regulate muscle strength during its development (25).

In addition, the patient reported a slight decrease in his upper visual field (despite the computerized visual field test showing normal results), which is likely related to spatial and movement perception, as assessed by the Test of Visual Perception Skills (TVPS). Most reports on the visual rehabilitation impacts of TBI do not measure visual perceptual skills. We assessed and rehabilitated the patient's awareness of the affected peripheral visual field (Table 1). Visual field awareness is a cognitive function that can also be enhanced by neurotrophic factors stimulated through voluntary exercise (23). To improve the visual field in patients with brain injuries, the most effective strategies include visual scanning training, body awareness techniques, and optical devices such as prisms (26).

Therefore, it can be hypothesized that the motor activities included in vision therapy can increase neurotrophic factor production, as occurs in voluntary exercise. However, studies are needed to measure the real effect of vision therapy on neurotrophic factors production and compare it with other treatment options.

CONCLUSIONS

Optometric vision therapy plays a fundamental role in the interdisciplinary management of patients with TBI, as demonstrated by the improvement in motor, sensory, and perceptual visual skills documented in the present case. The integration of visual and motor exercises, combined with a single botulinum toxin injection, not only led to a significant reduction in symptoms but also contributed to the sustained maintenance of these skills over time, positively impacting the patient's daily activities.

The success of the rehabilitation could be related to the increased production of BDNF through these controlled exercises. However, measuring the endogenous production of these neurotrophic factors induced by visual therapy is necessary to accurately evaluate this hypothesis.

Furthermore, additional physiological studies are needed to clarify the mechanism of action of optometric vision therapy, as it may reduce or even eliminate the need for repeated Botox applications

in patients with TNP caused by brain trauma. True multidisciplinary approaches are essential to maximizing therapeutic benefits and enhancing patients' quality of life.

Take-Home Points

- Optometric visual therapy could be a non-invasive option to recover sensory, motor, and perceptual functions affected in TNP secondary to TBI. Therefore, it should not be replaced by another specialist.
- Interdisciplinary work between optometrists and ophthalmologists is essential for treating patients with third nerve involvement secondary to TBI.
- Demonstrating that optometric vision therapy could be related to increased production of BDNFs through controlled exercise in experimental studies could be a major advance and support for treating TNP secondary to TBI.

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PATIENT CONSENT

A written patient consent statement was obtained for the identifiable health information included in this case report.

CONFLICT OF INTEREST DISCLOSURE

None of the authors has reported a conflict of interest.

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AUTHOR CONTRIBUTIONS STATEMENT

López Zapata Daniela: Project administration, Data curation, Writing - Original draft preparation, Conceptualization, Methodology. **Oyasa Moncayo Juan Fernando:** Writing - Review & Editing. **Medina Hernández Héctor Francisco:** Validation, Supervision.

REFERENCES

1. Bolton N, Gushchin A, Williams KM. Acquired third nerve palsy. Dis Mon. 2021 May 1;67(5):101131. https://doi.org/10.1016/j.disamonth.2021.101131
2. Roarty J. Third-Nerve Palsy - American Academy of Ophthalmology [Internet]. 2017 [cited 2025 Jan 6]. Available from: https://www.aao.org/education/disease-review/third-nerve-palsy-2
3. Aguirre-Castañeda CA, Benítez-Mejía JF, Morales-Cuellar J, Restrepo-López JS. Parálisis del III par craneal con disfunción externa completa y preservación pupilar como manifestación de aneurisma. Reporte de caso. Rev Ecuat Neurol. 2020;29(1). https://doi.org/10.46997/revecuatneurol29100145
4. Thomaz YB, Fernandes BA, Dantas EK de O, Lovato Júnior M. Paralisia total do III par craniano após trauma cranioencefálico: relato de caso. Braz J Health Rev [Internet]. 2024 Sep 27;7(5):e73155. https://doi.org/10.34119/bjhrv7n5-283
5. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Elsevier [Internet]. 2007 [cited 2022 Oct 12]. Available from: https://www.sciencedirect.com/science/article/pii/S1529183906008025
6. Chen N, Liao M, Yang C, Liu L. Accommodation and stereopsis in adults with traumatic brain injury. Clin Exp Optom [Internet]. 2020 Nov 1 [cited 2025 Jan 5];103(6):877–84. https://doi.org/10.1111/cxo.13056
7. Reza Khalili M, Roshanshad A, Vardanjani HM. Botulinum Toxin Injection for the Treatment of Third, Fourth, and Sixth Nerve Palsy: A Meta-Analysis. J Pediatr Ophthalmol Strabismus. 2024 May 1;61(3):160–71. https://doi.org/10.3928/01913913-20231120-02

8. Méndez Sánchez T de J, Oest Shirai LR, González Blanco Y, Castro Pérez PD, Zazo Enríquez RM. Diplopía binocular por paresia o parálisis oculomotoras: resultados de las opciones de tratamiento. Rev Cubana Oftalmol [Internet]. 2022 [cited 2025 Jan 5]. Available from: http://scielo.sld.cu/scielo.php?script=sci_arttext&pid=S0864-21762022000100008
9. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME. Vision therapy for oculomotor dysfunctions in acquired brain injury: A retrospective analysis. Optometry. 2008 Jan;79(1):18–22. https://doi.org/10.1016/j.optm.2007.10.004
10. Kapoor N, Ciuffreda KJ. Vision Disturbances Following Traumatic Brain Injury. Curr Treat Options Neurol. 2002;4:271–80. https://doi.org/10.1007/s11940-002-0027-z
11. Ciuffreda KJ. The scientific basis for and efficacy of optometric vision therapy in nonstrabismic accommodative and vergence disorders. Optometry [Internet]. 2002 Dec [cited 2025 Jan 5];73(12):735–62. Available from: https://pubmed.ncbi.nlm.nih.gov/12498561/
12. Barton JJS, Ranalli PJ. Vision therapy: Occlusion, prisms, filters, and vestibular exercises for mild traumatic brain injury. Surv Ophthalmol. 2021 Mar 1;66(2):346–53. https://doi.org/10.1016/j.survophthal.2020.08.001
13. Hernández Sarduy R, López Peña LI, Eirín Rey JE, Cruz Sáez HG. Rehabilitación oftalmológica de una parálisis del tercer par craneal como secuela de un herpes zóster ocular [Internet]. [cited 2025 Jan 5]. Available from: https://orcid.org/0000-0003-0900-6460
14. Caloroso E, Rouse MW, Cotter SA. Tratamiento clínico del estrabismo [Internet]. Madrid: Ciagami; 1999 [cited 2022 Jun 15]. p. 221–351. Available from: https://www.oftalmologos.org.ar/catalogo/items/show/3204
15. Urbanski M, Coubard OA, Bourlon C, Urbanski M, Urbanski M. Visualizing the blind brain: Brain imaging of visual field defects from early recovery to rehabilitation techniques. Front Integr Neurosci [Internet]. 2014 Sep 30 [cited 2025 Jan 6];8:94845. https://doi.org/10.3389/fnint.2014.00074
16. Przekoracka-Krawczyk A, Michalski A, Wojtczak-KwaAAniewska M. Visual Therapy in Open Space Rehabilitation of Acquired Visual Field Defect. Neuropsychiatry. 2018;08(05):1716–24. https://doi.org/10.4172/Neuropsychiatry.1000487
17. Griesbach GS, Hovda DA, Molteni R, Wu A, Gomez-Pinilla F. Voluntary exercise following traumatic brain injury: Brain-derived neurotrophic factor upregulation and recovery of function. Neuroscience. 2004;125(1):129–39. https://doi.org/10.1016/j.neuroscience.2004.01.030
18. Nudo RJ. Recovery after brain injury: Mechanisms and principles. Front Hum Neurosci. 2013 Dec 24;7:887. https://doi.org/10.3389/fnhum.2013.00887
19. Mellow ML, Goldsworthy MR, Coussens S, Smith AE. Acute aerobic exercise and neuroplasticity of the motor cortex: A systematic review. J Sci Med Sport. 2020;23(5):408–14. https://doi.org/10.1016/j.jsams.2019.10.015
20. Chae CH, Jung SL, An SH, Park BY, Kim TW, Wang SW, et al. Swimming exercise stimulates neurogenesis in the subventricular zone via increase in synapsin I and nerve growth factor levels. Biol Sport. 2015;31(4):-309–14. https://doi.org/10.5604/20831862.1132130
21. Carro E, Trejo JL, Busiguina S, Torres-Aleman I. Circulating insulin-like growth factor I mediates the protective effects of physical exercise against brain insults of different etiology and anatomy. J Neurosci [Internet]. 2001 Aug 1 [cited 2023 Jul 6];21(15):5678–84. https://doi.org/10.1523/JNEUROSCI.21-15-05678.2001
22. Liu H, Tan N, Xu D, Li CY, Xian GJ. NGF and CNTF expression and regulation mechanism by miRNA in acute paralytic strabismus. Int Ophthalmol [Internet]. 2020 Apr 1 [cited 2023 Jul 6];40(4):975–84. https://doi.org/10.1007/s10792-019-01270-x
23. Bastioli G, Arnold JC, Mancini M, Mar AC, Gamallo-Lana B, Saadipour K, et al. Voluntary Exercise Boosts Striatal Dopamine Release: Evidence for the Necessary and Sufficient Role of BDNF. J Neurosci [Internet]. 2022 Jun 8 [cited 2023 Jul 6];42(23):4725–36. https://doi.org/10.1523/JNEUROSCI.2273-21.2022
24. Ikeda K, Klinkosz B, Greene T, Cedarbaum JM, Wong V, Lindsay RM, et al. Effects of brain-derived neurotrophic factor on motor dysfunction in wobbler mouse motor neuron disease. Ann Neurol [Internet]. 1995 [cited 2023 Jul 9];37(4):505–11. https://doi.org/10.1002/ana.410370413
25. Chen J, Von Bartheld CS. Role of Exogenous and Endogenous Trophic Factors in the Regulation of Extraocular Muscle Strength during Development. Invest Ophthalmol Vis Sci. 2004 Oct 1;45(10):3538–45. https://doi.org/10.1167/iovs.04-0393
26. Love K. Visual-Spatial Therapeutic Rehabilitation for the Brain Injured Patient. Optom Vis Perform. 2014;2(3).