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A Cross-sectional study on Amblyopia Management

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KEYWORDS

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Abstract

Children's monocular vision loss is usually linked to amblyopia. To prevent vision loss, early detection and therapy are essential. Amblyopia is a complex developmental cortical situation of the visual pathway it is mainly brought on by defective visual the signal reaching the binocular the cortex cells. Parvocellular and magnocellular ganglion cells, the initial stage in the process of transformation of light energy into impulses from nerves, are the two types of ganglion cells. Magnocellular cells are engaged in gross stereopsis and movement perception, whereas parvocellular cells are involved in fine stereopsis, color vision, and visual acuity. When they occur during the critical time, strabismus, refractive error, cataracts, and ptosis are all highly amblyogenic. From birth to seven to eight years old is the important phase. Amblyopia treatments include of patching, atropine eye medications. Two hours of daily patching is just as effective as six hours is for children with moderate amblyopia, and daily atropine is just as effective as daily patching. The majority of the benefits of treatment are seen in children under the age of seven, while older children may also gain advantages. In 25% of kids, amblyopia recurs, thus ongoing monitoring is crucial.

Introduction:

Amblyopia, which results from aberrant vision development in infancy and early childhood, is a decline in best-corrected visual acuity **(Bradfield, 2013)**. The term, which is also known as "lazy eye," is derived from a Greek word that means dullness of vision. According to estimates ranging from 1% to 6%, amblyopia is the primary cause of monocular vision loss in children and causes irreversible vision loss in 2.9% of adults **(Attebo et al., 1998)**. Amblyopia is often unilateral, though it can occasionally be bilateral. Physical examinations typically reveal that the ocular structures are normal. Uneven refractive error and strabismus, or misaligned eyes, are associated conditions **(Bradfield, 2013)**. Conditions like cataracts and eyelid ptosis, which alter the visual axis and make it difficult to see clearly, can cause amblyopia. Bilateral amblyopia in children with significant refractive error is possible. In most cases, corrective glasses won't improve vision once amblyopia has developed. To prevent vision loss, it is crucial to identify and refer patients as soon as possible throughout infancy and youth.

Premature delivery, small for gestational age, developmental delay **(Mohan et al., 2001)**, being small for gestational age, **(Herbison et al., 2013; Li et al., 2013; Spiegel et al., 2013)**, or having a first-degree relative with amblyopia **(Repka et al., 2010; Dadeya et al.,2009)** are risk factors for amblyopia. According to certain research, environmental factors, such as maternal substance usage during pregnancy, enhance the chance of amblyopia or strabismus.

Amblyopia is believed to develop in infancy and the early years of childhood, when the integration of the visual cortex and the eyes is crucial for the development of vision. The visual brain becomes underused as a result of insufficient stimulation of the visual pathways, which causes amblyopia. Strabismic, anisometropic, and a combination of these sorts are the most prevalent types. Ametropic and deprivation amblyopia are less frequent types. When the eyes are not aligned, strabismic amblyopia develops. Because the brain cannot combine the disparate images from each eye, the deviating eye's visual input is suppressed by the visual cortex. Binocular vision is lost as a result of this type of amblyopia, and with it, stereopsis. When there is a discrepancy in visual acuity between the eyes, it can cause anisometropic amblyopia, also known as refractive amblyopia, which causes blurred vision in one eye. Because it takes more work for the eye with

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the larger refractive error (more blur) to focus and generate a sharp image, it frequently remains unfocused.

Types of Amblyopia

1. Deprivation amblyopia

When eye conditions prevent the light stimulus from reaching the retina, deprivation occurs, impeding the regular visual process. It may also be brought on by abnormal eye movement problems, such as nystagmus, or anatomical deficiencies of the retina or optic nerve. It can lead to amblyopia if it happens at the crucial stage of visual development. Amblyopia can also be brought on by other disease processes, including congenital cataract, blepharoptosis, nystagmus disorders, optic nerve coloboma and hypoplasia, retinal problems, and persisting fetal vasculature.

The first sort of amblyopia studied in the 1950s was that brought on by deprivation, according to works by Hubel and Wiesel. The authors showed that depriving cats' eyes of visual cues by suturing their eyelids caused several morphological and functional alterations in the cortical visual circuits. According to the authors, these alterations became more extreme the earlier, more intense, and the longer the deprivation lasted **(Tychsen, 2007; Tychsen et al., 2008).** Studies have also been done on the results of late eye closure. As a result, when closure took place after 10 weeks of age, the effect of deprivation on the size of the bands of the cortical ocular dominance columns was significantly diminished **(Headon, 1985).**

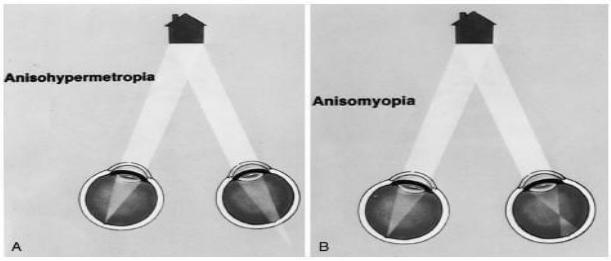
Therefore, the essential time of cortical alterations in a monkey would stop around 3 months, which would be equivalent to roughly 18 months of life in a human **(Barrett et al., 2004)**. The essential period of development has not yet concluded at this age, and the visual system is still open to various modifications **(Headon, 1985).** Several authors warn that despite the significance of results from animal models, comparisons between these models and human models should be thoroughly examined. Not only are there differences in brain structure between species, but deprivation is also recognized and controlled in animal models, but in the majority of instances involving children, there will be a variety of clinical symptoms and other related factors with amblyopia **(Barrett et al., 2004).** Taking this into consideration, numerous authors have demonstrated that deprivation has varying effects on children's vision and that the length of time and intensity of the deprivation will result in varying deficits in the ultimate visual function **(Lewis and Maurer, 2005; Ellemberg et al., 2000)**.

2. Anisometropic amblyopia

Anisometropia is a discrepancy of at least one diopter in the state of refraction between two eyes. About 4.7% of children have anisometropic amblyopia, which can be myopic, astigmatic, or hypermetropic. The most typical type of anisometropia appears to change according on the sample's age, ethnicity, and ocular diseases **(Huynh, 2006; O'Donoghue et al., 2013)**.

The most likely type of anisometropia to result in amblyopia is hypermetropic anisometropia, as the retina of the more ametropic eye never receives a sharp image: The healthy eye's fovea is focused, thus no accommodative effort will be stimulated to change the focus of the more hyperopic eye. The more ametropic eye can be employed for close vision in myopic anisometropia, preventing the same degrees of amblyopia as seen with hyperopia **(Toor et al., 2018)**.

Since the more ametropic eye is deprived of getting a high-quality retinal input, anisometropia may be regarded as a moderate sort of visual stimulus deprivation. Amblyopia caused by anisometropia is therefore expected to undergo anatomical and functional alterations similar to those seen in deprivation **(McKee et al., 2003; Levi et al., 2011)**.



Both deprivation and anisometropia cause a partial "disconnection" of the damaged eye from the primary visual brain, which causes aberrant neural competition. The proportion of cortex neurons responding to stimuli of the damaged eye is substantially lower in animals that have had one or both eyes blocked or blurred, compared to normal animals where most cortical neurons respond to stimulation of both eyes. Additionally, anisometropia and deprivation show a decrease in brain acuity. To put it another way, cortical neurons that are still sensitive to inputs coming from the damaged eye have receptive fields that are generally diffuse and insensitive, which results in worse spatial

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resolution and contrast sensitivity **(Movshon et al., 1987; Sengpiel et al., 1996)**. The quantity of anisometropia between the two eyes, rather than the magnitude of the refractive degree itself, determines how severe the amblyopia is.

3. Strabismic amblyopia

It is brought on by the eye's deviation. In this instance, the brain is presented with two distinct images, each with a distinct spatial projection, one of which is from the squinted eye. As a result, there is a neuronal vulnerability since the brain is unable to combine the images from the two eyes to produce stereoscopic vision.

When the visual system is in its critical period of development (in childhood), the brain is still able to use mechanisms to avoid diplopia or rivalry by inhibiting the activation of the retinocortical pathways originating from the fovea of the deviating eye. When the visual system is fully formed (when a person reaches adulthood), the perception of non-corresponding images by 2 eyes results in double vision. This adaptive mechanism prevents diplopia, but it reorganizes the visual cortex's cortical networks, which leads to amblyopia.

Although the cortical cellular infrastructure remains largely unaltered, the visual system undergoes numerous functional alterations. Retinal connection is fully disrupted, there is active and profound suppression of the dominant eye over the deviating eye, and cellular relationships are changed.

In addition to the loss of V1 binocular connections, Tychesen and colleagues have demonstrated other visual function abnormalities in strabismus-affected monkeys **(Tychsen, 2004)**. As the decorrelation time rose, so did the severity of the motor ocular alterations and the loss of V1 binocular connections. These functions were restored in the animals after just three weeks of decorrelation. Even after the deviating eye's position is corrected, other studies have shown that excitatory interactions for the deviating eye remain deactivated, but inhibitory ones do not **(Sengpiel et al., 1995; Sengpiel, 1996; Smith et al., 1997)**. This indicates active cortical suppression and an imbalance between the cortical cellular columns.

Strabismus alters the spatial summation and side inhibitions of incoming stimuli, which prevents the integration of contours and shapes. It also causes alteration in or loss of connectivity to the cortical spatial information pathways. Numerous selective visual tasks, such as visual acuity, Vernier visual acuity (alignment accuracy), and crowding, are affected by a distortion of the spatial vision **(Hess et al., 1978).**

There is no binocular facilitation for any sort of input in strabismus; instead, there is a continual, powerful suppression that is likely a modified version of the suppression of binocular rivalry. When the amblyopic eye is fixing, suppression is also detected in the normal eye's fovea, demonstrating that the cause of decreased visual acuity is not entirely suppression. Because the system may become inactive due to strabismus, this suggests that suppression rather than the other way around causes amblyopia in strabismus patients **(Sengpiel, 1996)**.

4. Mixed amblyopia

When two amblyogenic variables contribute to amblyopia, it is deemed mixed amblyopia. Especially in partially accommodative esotropia, microtropia, and monofixation syndrome, combination of anisometropic and strabismic amblyopia is prevalent **(Weakley, 2001).** Clinically, mixed amblyopia is more severe with comparable visual function impairments; visual acuity loss, contrast sensitivity, and stereopsia are frequently extinguished. The timing or concurrent start of each ocular change will determine when and how much of an influence it will have on each visual function.

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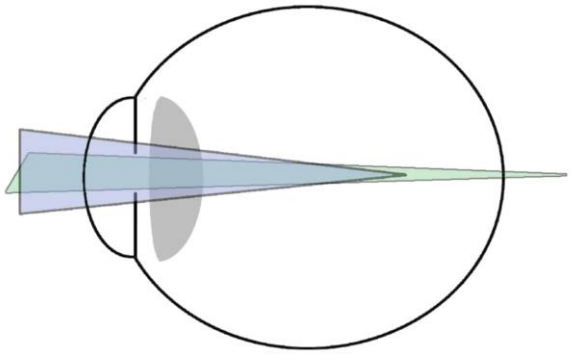

Screening and Diagnosis of Amblyopia

For the best chance of a satisfactory outcome from treatment, amblyopia must be diagnosed as soon as possible **(Kirk et al., 2008)**. According to **Holmes et al. (2011**), younger children are more likely than older kids to respond well to treatment. The American Academy of Family Physicians supports the U.S. Preventive Services Task Force's (USPSTF) recommendation that all children between the ages of three and five should undergo at least one screening for amblyopia or associated risk factors.9 The USPSTF concluded that there is insufficient proof that screening children under the age of three results in better vision outcomes. Beginning screening in infants is advised by the American Academy of Pediatrics, the American Association for Pediatric Ophthalmology, and Strabismus **(American Academy of Pediatrics, 2016).** As soon as a risk factor is identified, children with a higher risk of amblyopia should be referred for an ophthalmologic examination **(American Academy of Ophthalmology, 2017)**. Ptosis, birth weight less than 1,500 g (3 lb, 5 oz), cerebral palsy, disorders involving the eyes (such as Down syndrome), and a family history of amblyopia or strabismus are some of these.

Treatment of Amblyopia

Prevention

The identification of amblyopia risk factors requires vision screening **(Scheiman et al., 2005; Donahue, 2013)**. The possibility of preventing amblyopia increases with earlier detection and treatment of strabismus and clinically severe refractive error **(Williams, 2002)**. Although improvement in visual acuity can be reasonably anticipated in older children and teenagers, it appears that the potential for successful treatment is greatest in young children when amblyopia is present **(Eibschitz et al., 2000; Kvarnström et al., 2001; US Preventive Services Task Force, 2011**). Approximately three-quarters of children under the age of seven showed improvement in their amblyopic eye's visual acuity to 20/30 or better six months after starting treatment, according to a study by the Pediatric Eye Disease Investigator Group on the treatment of moderate strabismic and/or anisometropic amblyopia **(Glaser et al., 2002)**.

Choice of therapy

Age affects how well amblyopia treatments work **(Mohan et al., 2004; Holmes et al., 2011)**. But regardless of age, everyone should be able to receive care. The likelihood of recovering normal vision in an amblyopic eye relies on a number of variables, including the age at which amblyopia first appeared, its etiology, severity, and duration, its prior treatment history and how well it responded, how well the patient followed treatment instructions, and any coexisting disorders. The foundation of the treatment plan is addressing the root causes of amblyopia, correcting refractive errors, and encouraging the use of the amblyopic eye over the healthy eye. The aim of treatment is to achieve equal visual acuity in the two eyes, which is not always possible. The course of treatment should be determined by the child's age, visual acuity, compliance with prior therapy, response to that therapy, as well as his or her physical, social, and psychological well-being. Children's amblyopia is treated in the following ways:

- Optical correction of significant refractive errors
- Patching
- Pharmacological treatment
- Refractive surgery
- Alternative therapies

Optical correction

Children 0–17 years old with amblyopia are initially treated solely for refractive error **(Eibschitz–Tsimhoni et al., 2000)**. For patients with one eye having better visual acuity than the other, refractive error repair and compliance with the refractive correction pose challenges because many people with this type of anisometropic or ametropic amblyopia refuse to wear glasses. When wearing glasses consistently is problematic, refractive surgery can successfully fix the problem and improve vision **(Repka et al., 2005)**.

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When children's vision does not improve with eyeglasses alone, patching is started **(Repka et al., 2003)**. According to the Amblyopia Treatment Study (ATS), treating severe amblyopia (20/100 to 20/400) in children under the age of seven with 6 hours of prescribed daily patching results in an improvement in visual acuity that is comparable in size to full-time occlusion therapy. When children with moderate amblyopia (20/40 to 20/80) get the initial therapy of 2 hours of prescribed daily patching, their visual acuity improves to a degree comparable to that of 6 hours of daily patching. The patching-achieved therapeutic advantage seems to last for at least 15 years. For older kids and teenagers, patching should be taken into consideration, especially if they have never received treatment before **(Scheiman et al., 2005)**.

Pharmacological treatment

Children who do not improve with eyeglasses alone or whose compliance to patching is low for a variety of reasons, the existence of latent nystagmus, or maintenance therapy may be candidates for pharmacological treatment that results in cycloplegia of the nonamblyopic eye **(Repka et al., 2004)**. When the nonamblyopic eye is hyperopic, it functions best. The nonamblyopic eye is optically defocused by the cycloplegia. Through the age of 15 years old, the benefit of pharmacologic treatment is still steady **(Repka et al., 2014)**. The fellow eye has been given pharmacological treatment utilizing a range of dose plans. For initial treatment, it has been demonstrated that daily dose is just as effective as patching. For the treatment of moderate amblyopia over the course of four months, atropine 1% given twice weekly on consecutive days was just as effective as atropine 1% administered once daily. Children aged 3 to 12 with severe amblyopia showed a modest improvement of 4.5 lines (95% CI, 3.2-5.8 lines) with twice-weekly dosage **(Repka et al., 2009)**. Children who have stopped progressing with atropine 1% may have a slight benefit by adding a plano lens over the hyperopic fellow eye to their atropine therapy **(Wallace et al., 2015).**

Citicoline

Both cholinergic and neuroprotective effects are provided by citicoline. Initial research in adult patients showed that augmenting patching with citicoline improved VA, but that benefit was not maintained after stopping the medication. Early trials in children with amblyopia were encouraging, demonstrating the effectiveness of patching and citicoline alone as a therapy. An added citicoline group demonstrated a substantial treatment effect after 90 days in a study of patients who had never received treatment and were randomly assigned to receive it following a run-in patching period. Results from this study should be cautiously interpreted because the control group's failure to show improvement (2 hours per day of patching) was unexpected. At the time of this study, all studies on the usage of citicoline had not included follow-up periods longer than 3-6 months **(Campos et al., 1995; Fresina et al., 2008)**, suggesting that research on the drug is arguably behind that on levodopa.

Clinical Case Study

Clinical Case 1

A 7-year-old male patient come on my clinic with the complained of diminished of vision in his right eye. On examination his vision in right eye was 6/60 and in left eye was 6/6p in Snellen’s chart, Correction with glasses in right +3.50 Diopter, spherical (Dsph) and vision was improved 6/36p and in left eye correction with glasses +0.25 Dsph, vision was improved 6/6. After refraction under cycloplegic drugs like Homide, vision was 6/36 in Right with +4.50 Dshp, and 6/6p in left eye with +0.75 Dsph refractive errors.

On examination on slit lamp there is all are normal in parameters. There was no any abnormality in the Ocular Movements. Fundus was normal, Disc was hypermetropic, Intra ocular Pressure was 17 & 15 mmHg in right & left eye. So, diagnose was Anisometropia Amblyopia. I suggested him for the occlusion therapy (Patching) 6:1 ratio that means occlusion of left eye for 6 days and occlusion of right eye for 1 day. After 2 months examination of eyes, the vision of right eye improved 6/36 to 6/18 partial (p) with same correction in right eye and in left eye improved 6/6p to 6/6, so we continue same exercise for next 2 months. After 2 months period his visual acuity improved by 6/18 to 6/12p with correction of +4.00 Dsph. After 4 months spectacle number decrease around +0.50 Dsph (from +4.50 Dsph to +4.00 Dsph) and we change occlusion ratio by 3:4 (occlude right eye for 3 days and left eye for 4 days) and revive after 3 months. After 3 months vision 6/12 or 6/9p and patient has no diplopia.

Clinical Case 2

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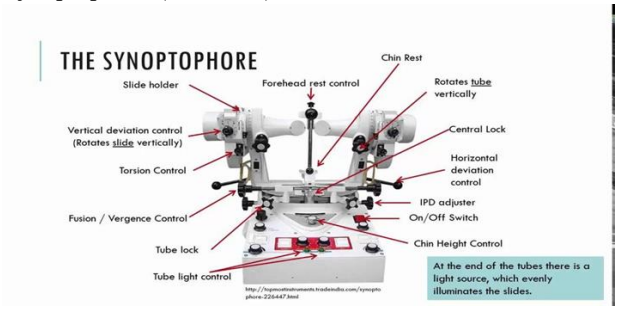
A 14 years old female patient come on my clinic with chief complained of diminished of vision in both eyes. When check her vision was 2/60 in right eye and 6/12 in left eye. After best correction right eye -9.0 Dsph vision was 6/36 and in left eye -0.75 Dsph was 6/6 p.

On examination on slit lamp there is all are normal in parameters. There was no any abnormality in the Ocular Movements. Fundus was myopic, disc was myopic, and Intra ocular Pressure was 18 & 14 mmHg in right & left eye. Now doctor gave her occlusion therapy 5:2 in ratio for 2 months and vision was increased 6/24 in right eye and 6/6 in left eye. After 2 months, the patient refraction was changed from -9.0 Dsph to -10.0 Dsph in right eye, and in left eye refraction was same as previous. After refraction the patient vision has been improved from 6/24 to 6/18 so suggested that continue the same occlusion therapy for the next three months. After completing three months of occlusion therapy the refraction has been changed from -10.0 Dsph to -10.50 Dsph and vision of right eye was improved from 6/18 to 6/12p, and in left eye refraction has been changed from -0.75 Dsph to -1.0 Dsph and vision was improved 6/6. After this therapy patient has been reported as diplopia so advised her to use the contact lens.

Exercise given by: -

- Synaptophore (HB Slide)



- Cam Stimulator

- Drawing
- Writing
- Lasy Eye Exercise
- Separation of Multiple Beans
- Playing Out Door

. U-Tube Exercise

Conclusion

The primary cause of amblyopia is aberrant visual signal that reaches the binocular cortex cells, which may be multimodal. Amblyopia is a developmental cortical abnormality of the visual pathway. Early detection and rapid treatment may lower the prevalence of amblyopia with screening performed before the age of 2 to 3 years. While the age at which treatment is initiated is not a factor in treatment success, early detection and implementation of treatment are key to improving amblyopia results. 63–83% of patients have reported receiving a successful course of treatment. Anisometropic amblyopia and mild occlusion may be successfully treated with refractive surgery alone, and atropine penalization may initially improve vision and encourage adherence to long-term therapy.

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