MEDWIN PUBLISHERS
Committed to Create Value for Researchers

Open Access Journal of Ophthalmology
ISSN: 2578-465X

# Recent Advances in Management of Amblyopia

**Gupta SB[1]*, Kashiv Y[2] and Khan N[3]**

[1]Mahaveer Institute of Medical Sciences and Research, India

[2]SR People medical college, India

[3]Asst professor, MIMS India

***Corresponding author:** Swarna Biseria Gupta, Mahaveer Institute of Medical Sciences and Research, d-5 Machna colony, Jabalpur, Bhopal, India, Tel: 09425607342; Email: biseriaswarna4@gmail.com

**Mini Review**

**Volume 8 Issue 1**

**Received Date:** March 16, 2023

**Published Date:** April 28, 2023

DOI: 10.23880/oajo-16000267

## Abstract

Amblyopia is the most common cause of monocular vision loss in children. Global incidence is 3.7%. It may be due to early deprivation of vision or defocus in one eye, reflects an imbalance of input from the eyes to visual cortex .Not only monocular vision is reduced but binocular vision is also affected. Fellow Human brain is designed to allow both eyes to work together. If signals from one eye are blurred, brain blocks the visual input from that eye. In the visual pathway, the synapses are broken due to disuse of amblyopic eye. Along with visual coordination deficit, eye to hand and reading can be affected and self-perception may be diminished. A variety of amblyopia therapy options have been developed to treat amblyopia in children and adults. Amblyopia therapy options have traditionally been limited to penalization of non-amblyopic eye with either patching or medicinal penalization. Traditional approaches to diagnose and treatment have limited effectiveness, are uncomfortable for the patients and their families. Recent evidences shows that amblyopes possess binocular cortical mechanisms for both threshold and supra-threshold stimuli. Hence strategy may be based on binocular stimulation methods, aims to stimulate either eye leading to a concomitant improvement in monocular vision with reduction in suppression and strengthening of binocular vision.

**Keywords:** Amblyopia; Binocular Vision; Mono Ocular Vision; Penalization; Occlusion; Newer Strategies

## Introduction

Amblyopia is diminution of vision, not attributed to the structural abnormality of eye, described as amblyopia ex anopsia. The mechanism of retinal and cortical interaction varies in different conditions. The first mention of amblyopia dates back to the fifth century BC to the old Greek. Back then Hippocrates used the word amblyopia to name conditions that resulted in decreased visual acquity in seemingly healthy eyes. It occurs when there is a major difference between the two eyes in their ability to focus. Any child with visual acquity in either eye of 6/12 or less at the age of 3 to 5yrs or 6/9 [1] or less at the age of 6yrs or more, or two line difference in vision of two eyes, diagnosed as amblyopic. In addition to low vision, there is decrease in contrast sensitivity, vernier acquity, spatial distortion and low form sense [2]. An adult person with unilateral amblyopia has three times greater risk, while a child, 17 times that of normal person for decrease in vision in better eye. In strabismus, the foveae in two eyes are directed towards different visual objects, it may cause visual confusion, retinal rivalry, but no diplopia, because of faint image in amblyopic eye. Amblyopia is more common in premature infants, usually have poor prognosis. Critical period is time in early life, usually begins at 4 months of age, peak is at 2yrs and gradually decreases by 5yrs, then ceases at 12 yrs.

### Anatomy and Physiology of Retino-Geniculo Cortical Pathway

In amblyopia, the cortical changes are not limited to V1 and extend beyond. The critical period extends from birth to 7-8 yrs. Ocular morbidities, occurring during this period are highly amblyogenic. The normal visual stimuli is essential for cortical development during this critical period and this continues till the age of 7yrs and even after, and never lost before 50yrs [3]. The ganglion cell is the first step where light energy converts to nerve impulse, which is involved in fine visual acquity, stereopsis, color vision and movement recognition and P cells of ganglion cells have a larger representation in cortical areas and crossover to lateral geniculate body. Retinal fibers end in the lateral geniculate body in different layers [4].The axons of relay cells in the lateral geniculate nucleus terminate in layer 4 of primary visual cortex segregated over first 3weeks of life to form ocular dominance column.

Hence if one eye is closed at birth, its ocular dominance bands become narrow. If closure delayed to 6 months, shrinkage of cell bodies in LGN occurs, because monocular closure prevents binocular cooperation. The ocular dominance is amenable to alteration, maturity never lost till the age of 36 months postnatal. However cortical plastic is never lost fully in adults and by the ways of restoring plasticity, amblyopia may be treated. As brain is not a physiologically static organ and it can modify throughout life [4], it can be stimulated to form new connections between existing brain cells and old ones by strong, persistent and appropriate stimuli. The molecule responsible for neuroplasticity is a protein receptor, active brain stimulator, GABA [5], an excitatory neurotransmitter, develop and regulate proliferation of neural progenitor cells. Dopamine is another neurotransmitter, present in retina and cerebral cortex, stimulates receptors and turns them ”on” [6]. The concept of neuroplasticity shows that amblyopia can be treated in adults too.

## Pathogenesis

Amblyopia arises from lack of consistency between the images of each eye; information from one eye becomes more privileged. While the picture coming from the other eye is actively suppressed by the visual cortex [4]. Suppression leads to decreased visual acquity in this eye and disrupts binocular vision, which is a primary leading factor in the development of amblyopia. In amblyopic patients, there is deficit of processing of visual information at high levels of visual system, in the areas of parietal occipital and temporal cortex, revealed a reduction in the number of binocularly controlled neurons. It may cause suppression [2]. Regardless of the etiology of amblyopia, patients, who don’t have stereoscopic vision, possess deficient visual acuity in optotypes. Suppression inhibits information from the amblyopic eye to prevent diplopia and confusion. It affects many aspects of spatial vision; contrast sensitivity, hyper acuity, crowding effect [6]. Local temporal processes are less affected by amblyopia than local spatial processes.

## Etiology

The causes of amblyopia can be divided as

- Strabismus (50%)
- Anisometropia (17%)
- Strabismus+ Anisometropia (30%)
- Ametropia (3%)
- Sensory deprivation (3%)
- Retinal/optic nerve diseases (3%)

### Risk Factors

It includes

- Family h/o amblyopia
- Family h/o squint
- Pediatric cataract, glaucoma
- Premature birth
- Delayed neurological and visual idiopathic maturation
- Socioeconomic factors
- Refractive errors, visual deprivation in early childhood anisometropia of more than 1D in hypermetropes and 2.5D in myopes can cause amblyopia and decreased binocularity. Meridional amblyopia occurs if the astigmatism is more than 1.5 dioptres. Ametropic amblyopia occurs in children with hyperopia greater than 4.5 D and myopia more than 6D.

### Types

**Strabismic amblyopia**

- Misalignment of eye results in abnormal binocular interaction.
- Eventual unconscious suppression of visual stimulation to an affected eye creates amblyopia.

**Deprivation amblyopia**

- Eye fails to receive clearly formed image on retina.
- Due to a cataract, corneal opacity, ptosis, lid tumor.

**Refractive (Anisometropic)**

- Difference in refractive error between two eyes.
- Clearer image favored.
- Visual loss (blunting of vision) in eye with higher refractive error.

**Symptomatology**

- Frequent squint, rubbing of eye, closes one eye.
- Turns head to one side.
- Reading difficulties; skipping words, misreading, substituting words.

- Stressful reading and fatigue [6]

Sport performance difficulties due to faulty depth perception; frequent tripling, falling, bumping

Reduced fine motor skill; Messy handwriting, because of poor binocular coordination.

Attention difficulties due to reduced focus.

Defective vision, without any organic ocular morbidity.

## Diagnosis

### Tests for detecting Amblyopia

**Visual Acuity Tests**

- It is subjective and should be interpreted in relation of patient's age, ability and cooperation, by snellen's chart in grown up subjects or by naming, matching the letters or symbols on a chart fixed at particular distance [7].
- Visual acuity test, in preverbal infants and toddlers, done by assessing fixation using Teller's chart.
- Visual acuity, corrected with spectacles, power of lens used.
- Visual acuity with pin hole.

**Optical Tracking:** Amblyopia causes small abnormalities in fixation in amblyopic eye. Optical tracking is done to measure eye movements to detect small abnormalities in fixation.

**Optiko Kinetic Nystagmus:** It is a reflex, developed around 6months of life. It is horizontal rapid movement seen. On following a moving object, can be recorded by optiko kinetic drum [8].

**Natural Density Filter Testing:** It is used to identify amblyopia is due to strabismus or any other ocular morbidity [7].

**Visual Evoked Potential:** Changes in voltage measured with sensitive electrodes placing on scalp adjacent to occipital lobe, the VEP in vision screening is tested by showing, progressively small size objects, until VEP becomes extinguished.

### Test of Stereopsis and Binocular Vision

**External Examination:** For any lesion, this could affect visual development

- Anterior segment examination

For any opacity in media or irregularity

- Status of afferent pupil defect
- Motility and ocular alignment

**Fundus Examination:** Neuroimaging Techniques [9].

### Tests for Assessment of Risk Factors

**Photo screening:** It is the interpretation of photographs that reveal ocular defects, which may be analyzed to detect ocular pathology, refractive errors and opacity in media, strabismus and external eye abnormalities.

**Auto-refraction:** It is automated retinoscopy, determine refractive error in each eye [6]. Few recent auto refracto-meters provide sensitive screening for detection of amblyopia in children.

**Screening Programs:** Preschool screening: Periodical visual acuity screening by volunteers, camps organized by social clubs, pediatricians, community centers.

**School Screening:** By teachers, school surveys at base level, eye camps

### Aims of Amblyopia Therapy

- To provide a clear retinal image to amblyopic eye by correcting refractive errors.
- To assist patient, to use the amblyopic eye.

### Modalities for Treatment

**Refractive Correction**

**Guidelines**

- Refractive error estimation under cycloplegia
- Full correction irrespective of refractive error.
- hyperopia is fully corrected; in esotropia
- The time frame for VA improvement varies but can take upto 1yr or more with improvement in stereopsis.

**Occlusion:** When patient do not respond to optical correction, VA ceases to improve, occlusion may be done as therapy using an eye patch to cover the non-amblyopic eye for a couple of hours each day. The sensitive in which vision loss may be recovered is generally up to 6yrs of age [10]. It is the treatment of choice for amblyopia, success rate varies from30% to 92% [11]. Depends on patient selection, treatment duration and age of patient, compliance strategy and type of amblyopia.

**Guidelines**

- Full time occlusion provide excellent results; 88% patients achieved 6/6 visual acuity and maintained after 16yrs follow up [10].
- In children 4-6yrs, 6hrs occlusion, improve in moderate & severe amblyopia.
- For severe amblyopia, 6hrs of daily occlusion results an improvement similar to full time occlusion in children 3yrs to 7yrs of age.
- For moderate amblyopia, 2hrs occlusion daily produces same improvement, as in occlusion for 6 hrs daily in children from 3yrs to 7yrs.
- Problem with full time occlusion is disturbed routine, due to occlusion of sound eye.

**Penalization**

Penalization is used, when either occlusion does not work or post occlusion, as maintenance therapy [9].

**Indications**

- Moderate amblyopia in non-compliant patients
- Occlusion failure.
- Maintenance therapy
- Atropine penalization is used as an alternative to occlusion. It may be used alone or in combination with optical penalization (use of fogging lens over sound eye) [11].
- Benefit of atropine penalization is its easy administration, reliable compliance and low cost. Overcome social stigma and skin irritation due to patching. But its toxicity and prolong action are big problems, if reverse amblyopia is detected [11].
- Atropine or patching for 6 months period produces a similar improvement in amblyopia two years after treatment.

## Pharmacological Therapy

Combination of levodopa-carbidopa [12] in dosage of 0.95/0.24mgm and 1.94/0.49mgm/kg , improves visual functions in amblyopic eyes and found to be well tolerated. This combination produce two lines improvement in children younger than 8yrs of age. Citicoline (cytidine 5'-diphosphocholine) used in doe of1,000mgm IM for 15 days to the patients aged 9-37yrs (mean16.6yrs),produced a temporary improvement in visual acuity without any side effects [8].

### Near Visual Activities

Near visual activities are prescribed with occlusion to stimulate the visual system, by use of television games, mobile games and painting etc. It improves eye to hand coordination. Binocular game play is superior to monocular games for visual acuity and stereopsis [10] improvement. Dichoptic games are more useful.

### Role of Refractive Surgery

LASIK is a method for correcting high myopic and hypermetropic anisometropia.

### Acupuncture in Amblyopia

Acupuncture at vision related acupoints may modulate the activity of the visual cortex. It is effective in increasing blood flow to cerebral and ocular vasculature, stimulating retinal nerve growth factors [13].

### Trans-cranial Magnetic Brain Stimulation

It is noninvasive procedure used for stimulating parts of brain by use of weak electric current, induced by rapidly changing magnetic currents

### Role of Omega Fatty Acids

Omega fatty acid is important structural components of membrane lipids in central nervous system, and found beneficial for retinal maturation and visual acuity development.

### Newer Strategies

As amblyopia is a binocular problem caused by active suppression results in functionally monocular system. Hence binocular treatments, improve vision as well as binocularity.

### Principles

- Monocular perceptual leaning (PL)
- Monocular video game play (VGP)
- .Dichoptic PL/VGP

A Dichoptic treatment stimulates each eye separately and the brain is forced to integrate the images into a single perception. The signal strength coming to good eye is reduced so that it cannot suppress the amblyopic eye. Few software's are available to promote binocular vision therapy with 3D gaming, helpful to treat amblyopia not only in children but in adults too.

### Programmable Electronic Glasses

The lenses are liquid crystal display, programmed to turn opaque, occluding vision in either eye for different interval, acting like a digital patch that flickers on and off, as per requirement.

## Conclusion

Amblyopia, being the common cause of monocular vision loss with visual function deficits. In children and population under 40yrs, due to prolong abnormal retinal stimulation, needs improvement of vision in amblyopic eye, by using binocular stimulation procedures. Conventional Occlusion is still treatment of choice, preferably part time occlusion, penalization may be considered are as alternate, near vision exercises are useful, pharmacological therapy has limited role, because of its side effects.

## References

1. Ederer F, Krueger DE (1984) Report on the National eye institute's Visual acuity impairment survey pilot study. Washington pp: 81-84.

2. Banks MS, Aslin RN, Letson RD (1975) Sensitive period

for development of human binocular vision. Science 90(4215): 675-677.

3. Barrett BT, Bradley A, Graw PVM (2004) Understanding the Neural Basis of Amblyopia. The Neuroscientist 10(2): 106-117.

4. McKee SP, Levi DM, Movshon JA (2003) The pattern of visual deficit in amblyopia. J Vis 3(5): 380-405.

5. Holmes JM, Beck RW, Repka MX, Leske DA, Kraker RT, et al. (2001) The amblyopia treatment study, visual acuity testing Protocol. Arch Ophthalmol 119(9): 1345-1353.

6. Pediatric Eye disease investigator Group (2003) The course of moderate amblyopia treated with atropine in children. Am J Ophthalmol 136(4): 630-639.

7. Pediatric Eye disease investigator Group (2009) Pharmacological plus optical penalization treatment for amblyopia. Arch ophth 127(1): 22-30.

8. Repka MX, Kraker RT, Holmes JM, Summers AI, Glaser SR, et al. (2014) Atropine vs. patching for treatment of amblyopia. JAMA Ophthalmol 132(7): 799-805.

9. Birch EE, Li SL, Jost RM, Morale SE, Cruz ADL, et al. (2015) Binocular Ipad treatment for amblyopia in preschool children. J AAPOS 19(1): 6-11.

10. Dadeya S, Vats P, Malik KPS (2009) Levodopa/carbidopa in treatment of amblyopia. J Pediatr Ophthalmol Strabismus 46(2): 87-90.

11. Ari YB, Gaiarsa JL, Tyzio R, Khazipov R (2007) GABA: a pioneer transmitter that excites immature neurons. Physio Rev 87(4): 1215-1284.

12. Chakraborty R, Chatterjee A, Choudhary S, Chakraborty PK (2007) Neuroplasticity, a paradigm shift in neurosciences. J Indian medical assoc 105(9): 513-514.

13. Zarate BRD, Tejedor J (2007) Current concept in management of amblyopia. Newer strategies are better, in stimulating binocular interaction. Clinical Ophthalmology 1(4): 403-414.