Review Article

Seeing World From The Eyes of Low Vision Subject

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Abstract

Low vision is a moderate to severe form of visual impairment wherein best corrected visual acuity varies from less than 6/18 to 3/60 in better eye. A low vision subject is potentially able to use his residual vision for execution of tasks of his interest. Low vision may be due to retinal diseases, glaucoma and corneal affections. The visual and allied stimulus carrying neurons including photoreceptors and retinal ganglion cells get affected, resultantly the low vision subject has various forms of visual disabilities including diminution of vision, reduced contrast sensitivity, altered colour perception, defective depth perception, deficient form vision and glare. Therefore, affected subject sees the world differently from a normal one. The article describes how different aspects of vision are defeated due to low vision. Various rehabilitation measures including magnification, modified reading text material, increased lighting, visual stimulation and coloured lenses have been utilized in a pursuit to improve entire seeing experience of a low vision subject. However more extensive and detailed studies are required in order to assess the usefulness of these low vision aids in improving different aspects of visual functions.

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Introduction

According to the WHO Working Definition of Low Vision (1992), a person with low vision (LV) has an impairment of visual functioning even after treatment and/or standard refractive correction, and has best corrected visual acuity (BCVA) of less than 6/18 to 3/60 in better eye, or a visual field of less than 10^0 from the point of fixation, but who uses, or is potentially able to use, vision for the planning and/or execution of a task. In other words, LV is a "moderate to severe form of visual impairment."[1] In the world, there are about 36 million blind people with 217 million people having severe to moderate visual impairment for distance (i.e. LV) and 8.8 million blind with 47.7 millions having LV in India.[2,3]

Table: 1 showing category of visual impairment based on Best Corrected Visual Acuity (BCVA) of better eye ; Adapted from International Statistical Classification of Diseases and Related Health Problems, tenth revision, Geneva, World Health Organisation, 1982

Category	Corrected VA- better eye	WHO definition	Working	Indian Definition
0	6/6 – 6/18	Normal	Normal	Normal
1	<6/18 – 6/60	Visual impairment	Low vision	Low vision
2	<6/60 – 3/60	Severe visual impairment	Low vision	Blind
3	<3/60 – 1/60	Blind	Low vision	Blind
4	<1/60 - PL	Blind	Low vision	Blind
5	No PL	Blind	Total Blindness	Total Blindness

In India, the National Programme for Control of Blindness (NPCB) has been re-designated as National Programme for Control of Blindness and Visual Impairment (NPCB & VI). Further, under NPCB & VI, definition of blindness has been modified in line with definition given under the World Health Organization i.e. Blindness is defined as presenting distance BCVA less than 3/60 (20/400) in the better eye or limitation of field of vision to be less than 10^0 from centre of fixation, while earlier blind category under NPCB included VA <6/60 to 3/60 also.[4]

Causes Of Low Vision

The LV can be due to retinal diseases, corneal diseases or glaucoma. Few studies have been conducted to compare the incidence of LV from these diseases. In a study conducted in Turkish population, it was found that among those aged 18-50 years, retinal dystrophies (37%), congenital eye anomalies (14%) and myopic degenerations (13%) were the most common causes for LV. For those aged above 50 years, age-related macular degeneration (ARMD) (21%) was the leading cause. Diabetic retinopathy (17%), corneal opacities (14%), cataract (12%) and glaucoma (9%) were also important.[5]
In another study by Ackuaku-Dogbe et al, glaucoma (22.35%), retinitis pigmentosa (RP) (8.94%), ARMD (7.95%), non-glaucomatous optic atrophy (10.26%) were leading causes of LV in subjects attending teaching hospital at Ghana.[6]

Pathogenesis

The retinal diseases causing LV include retinopathy of prematurity (ROP), albinism, heredo-macular degeneration, pathological myopia and myopic retinopathy, RP, co-existing optic atrophy (Primary or secondary), ARMD, diabetic retinopathy and chorio-retinitis.
In ROP, the cell invasion of vitreous gel causes contractile force from cicatricial tissue leading to retinal detachment (Separation of neurosensory retina i.e. rods and cone from RPE) and subsequent degeneration of photoreceptors. In albinism, misrouting of the nerve fibers and abnormal foveal development occurs due to deficient melanin, while appropriate melanin has been included among several factors present during embryogenesis, which guide

the development of retinal and vascular structures and the routing of ganglion cells in the retina to the lateral geniculate nucleus and on to the occipital cortex. This results in reduced visual acuity. Other pigments, such as lutein and xanthophyll are not affected in albinism.[7] In heredo-macular degenerations, there is malfunction of the ATP-binding cassette transporter (ABCA4) protein of the visual phototransduction cycle leading to improper shuttling of vitamin A throughout the retina.[8] In pathological myopia, the axial elongation of the eye results in stretching of the ocular layers and progressive thinning of the retina and resultant cone-rod dysfunction.[9] In RP, there is a progressive bilateral degeneration of the rod and cone photoreceptors that leads to night blindness and progressive visual field defects. The optic atrophy is accompanied with loss of ganglion cells and axon degeneration in the retinogeniculate pathway (lateral geniculate body) leading to visual dysfunction.[10]

In ARMD, there is an oxidative damage to retinal pigment epithelium (RPE) cells, complement deposition in the RPE-Bruch's membrane-choriocapillaris complex, loss and later total disappearance of rods and cones at fovea and parafovea, vascular leakage from choroidal new vessels and subsequent reparative response resulting into disciform scars formation.[11,12] The diabetic retinopathy involves systemic microangiopathy, retinal plasma exudation and punctate haemorrhages, maculopathy due to cystoid oedema, closure of capillaries leading to cotton-wool spots and intra-retinal microvascular anomalies, rod and cone photoreceptor outer segment degeneration and retinal ganglion cell (RGC) loss.[13,14] The chorio-retinitis involves retinal and choroidal inflammation, vasculitis and photoreceptor destruction.

Development Of Clinical Presentation

In retinal diseases, the development of clinical presentation is due to affection of photoreceptors, bipolar cells and retinal ganglion cells. While cones confer chromatic vision, rods confer achromatic vision. Though both rods and cones are required for visual perception, cones are more closely linked to functions of VA, colour vision and contrast sensitivity as cone-mediated vision can detect a contrast (either spatial or temporal) as small as 0.5%, whereas rod-mediated vision needs a minimum contrast of 5% to be detected. Stereopsis appears to be processed in the visual cortex by fusing and processing images with disparity, but good VA is required for visual message "necessarily" to be sent to the cortex, which happens to take place when photoreceptors are healthy and functioning well. The glare is due to reduced amount of xanthophyll pigments including lutein and zeaxanthin, for example in ARMD, which otherwise filter some harmful blue light. This was found by Stringham and Hammond, who measured these pigments with Maxwellian-View Optical system and found reduced macular pigment optical density. The xanthophyll pigments also act as antioxidants to tackle free radicals and eradicate reactive oxygen species which can damage photoreceptors of macula.[15,16] In glaucoma, LV results from damage to the retinal ganglion cells which are the final visual message sending neuron via optic nerve to the visual cortex, while other layers of retina are minimally affected. The corneal diseases cause LV by interrupting appropriate light stimulus from reaching the photoreceptors preventing initiation of an ocular/visual stimulus.

Figure 1: *showing glare experienced by low vision subject*

Clinical Presentation

The LV subjects have glare (Figure 1) and this is more prominently seen in diseases like aniridia, ARMD and albinism. There is escapism from light and subject's face may have a downward tilt. He may need sunglasses, visors or hats outdoors as well as indoor.

The diminution of vision is the prime presenting clinical feature. The LV subject has difficulty in seeing distant and near objects. Hence he/she may go very close to the object in order to see it. Prescription of proper refractive correction for both near and distance is required. Additionally, magnifiers which are worn on glasses or hand held or in form of electronic devices which enlarge target of interest or enlarge the reading text are required. Goldstein et al found that though VA is the strongest predictor of visual ability ($P < .001$) and reading ability ($P < .001$), it additionally has a significant independent effect on the other functional domains including mobility, visual motor function, and visual information processing.[17] Thus subjects having diminished vision have difficulty not only in reading but it also reflects through reduction of their movement in their surroundings and carrying out daily activities like cooking food, washing clothes and cleaning their houses.

The LV subjects experience loss of colour vision and have an altered colour perception (Figure 2), thus they have difficulty in identifying food cooked, clothes to be worn, ripe fruits which are fit for consumption, etc. Jolly et al found that CVD deteriorates more and more as degeneration/disease keep progressing to encroach upon the fovea.[18]

As these patients experience a reduction in depth perception (Figure 3) and in recognising edges of objects, they have difficulty in performing daily activities like driving and reading; and children may find difficulty in learning for example through shapes of objects. The patients lose the

Figure 2: Showing *altered colour perception in low vision subject*

Figure 4: *Showing central field defect in a low vision subject*

Figure 3: *Showing reduced depth perception in a low vision subject*

Figure 5: *Showing reduced contrast sensitivity in a low vision subject*

ability to distinguish a relative physical distance between objects. Even monocular cues become ineffective thus it becomes difficult to appreciate the relative location of objects using healthier eye (in situation when the other eye has an extremely poor vision). As stereopsis is directly related to simultaneous perception from both eyes and good vision in both eyes, hence it is the level of VA in the poorer eye which defines the achievable level of stereopsis. Fine stereopsis is possible only in those who have good VA as well as good macular function in the poorer seeing eye also. However, only gross stereopsis is possible in those subjects who have a loss of macular function.[19] The studies have shown that binocular dysfunctions are prevalent in LV patients although stereopsis is present in about two thirds of tested subjects.[20,21]

The subjects who have central field defects (Figure 4) for example in heredo-macular degeneration, optic atrophy, ARMD and toxoplasma scar, turn their head while reading in order to use parafoveal/paramacular region for reading and use the same eccentric viewing to see distant objects. Patients with only central diseases however preserve peripheral fusion. Since peripheral fusion is as potent as central fusion, peripheral stereopsis may be present in LV cases, which is evidenced by the excellent spatial orientation and mobility skills noticed in many of such patients.

The LV subjects with wide impact of the disease process however may experience loss of both central and peripheral vision. For example, the concurrent peripheral field defects also occur in RP, lasered diabetic retinopathy and glaucoma. The subject resorts to eccentric viewing, head turn/tilt, tentative gait, maintenance of close proximity to walls or handrails and reliance on tactile information by holding onto individuals.

The LV subjects have a reduced contrast sensitivity (Figure 5). Resultantly, they have difficulty in driving, watching television and even reading text.

The form vision, which is recognition of visual elements of objects, specifically those to do with shapes/patterns/ previously identified important characteristics, is also disturbed in LV subject (Figure 6). This results from structural changes in retina involving displacement of both external retinal layers with improper light signal transduction and internal layers distortion from sub retinal neovascular membrane (SRNM). The disturbance in form vision may present as metamorphopsia.[22]

The patients with LV do not show a close relationship between threshold visual angle for isolated capital letters and for continuous text. As printed material presents a more complex and difficult task than do acuity letters, therefore

Figure 6: *showing reduced form vision in a low vision subject*

conventional tests of acuity are of little use in predicting magnification that the patient needs to read a continuous text with reasonable speed and accuracy. There is often a poor agreement between reading acuity and distance letter acuity and the discrepancies between these become more pronounced when there is a disturbance of macular function as in macular degeneration and amblyopia. So, assessing reading speed with use of objective tests like MN read test is required.[23]

Other presentation of LV may include nystagmus from poor vision and oculo-digital sign/eye poking sign (Leber's congenital amaurosis) from difficulty experienced in seeing. Loss of vision in one eye or both can have a profound effect on binocular visual functions. In most cases with LV, the fine balance of binocularity reached in the past is broken and emerging symptoms may disable many activities of daily living (ADL) as a result of binocular dysfunction.[24] The subject may have difficulty in daily living, soiled clothing and missing buttons. The overall appearance is a fatigued appearance which is indicative of ocular or systemic disorder.

Glaucoma

The LV due to glaucoma has somewhat different clinical presentation than LV from retinal diseases. Glaucoma causes damage to the optic nerve and retinal ganglion cells. It shrinks overall area seen and causes defects in side (peripheral) vision resulting into tunnel vision whereby central vision is preserved while peripheral vision is lost. In retinal diseases however, peripheral vision is generally preserved till later stages as central vision gets affected earlier from macular involvement. Tunnel vision of glaucoma is described as looking at the world through a straw or a pinhole and LV subject may not be able to see what is directly below, above or around him on the sides. People with glaucoma can often still see small details, but they lose the ability to see the entire picture. There is also difficulty with night vision, reduced ability to adapt from light to dark and from dark to light (for example coming out of a movie theatre at daytime), increased sensitivity to light (glare) and decreased ability to see contrast.

Corneal Diseases

The clinical presentation of low vision in corneal diseases has been infrequently described. Corneal opacity from congenital, nutritional, traumatic, infectious, degenerative, and hereditary conditions can cause LV. A corneal opacity can also result from poor quality cataract surgery and pterygium. In a Corneal Opacity Rural Epidemiological (CORE) study by Vashist et al, the VR-QoL (vision-related quality of life) was assessed through Indian Vision Function Questionnaire (IND-VFQ-33) in 435 adult participants (aged ≥18 years) having corneal opacity across three domains of vision function i.e. vision-specific mobility, psychosocial impact and visual symptoms and it was found that these subjects had significantly higher score and hence poorer VR-QoL than equal number of 435 healthy controls having VA of 6/6 in both eyes ($p<0.0001$). Additionally, the scores were inversely related with the level of visual impairment in patients with corneal disease. Patients with unilateral corneal disease also had poorer VR-QoL scores as compared with healthy controls ($p<0.0001$). The authors concluded that VR-QoL is impaired in patients with corneal disease, more so in patients with corneal blindness.

Clinical Examination

The traditional charts (e.g. Snellen's Chart) used in clinical practice are not standardized; they have an irregular progression of letter sizes and a variable number of characters per line. Measurement accuracy may further suffer from hidden errors that cannot be captured by any recording device- such as inconsistent and non-standardized protocol including viewing distance, inaccurate projector adjustment and contrast loss from room illumination. Today, the ETDRS chart and ETDRS protocol, established by the National Eye institute in the US, are considered to represent the de facto gold standard for VA measurements. The International Council of Ophthalmology, Visual Standard, Aspects and Ranges of Vision loss (April 2002) is a good reference document.[25] The Early Treatment Diabetic Retinopathy Study (ETDRS) distance and near are standard tests for examining LV subjects. The distance BCVA is tested on self-illuminated ETDRS acuity charts under uniform room illumination at 4 m. The ETDRS acuity log score which patient can read

completely is noted. Therefore, the difference between two routine clinical measurements should not be considered significant, unless it exceeds 1 line on an ETDRS chart.[26]

The use of conventional acuity, especially the distance value has, however, been criticized for not being ideal to calculate near VA and for reading power calculation. There is often a poor agreement between reading acuity and distance letter acuity and these discrepancies become most pronounced when there is disturbance of macular function as in macular degeneration and amblyopia. Thus the subjects' near vision is tested using ETDRS N charts having text printed in high contrast, at reading distance of 40 cm, using optimal illumination with recommended glass prescription. Same reading material having same contrast, illumination, letter spacing and font style is used in all subjects. The VA values measured at reduced near viewing distance from ETDRS near vision charts are used for predicting optical powers and visual performance of LV patients.

The colour vision assessment may be done by most commonly available Ishihara's tests but since the LV subjects suffer from undefined colour vision defect (CVD) thus Farnsworth D15/100- hue test is the most recommended one. In these tests, coloured discs are arranged in apparently correct order according to gradation of hue by LV subject in an ambient illumination, and variation from normal pattern (diagram on booklet) provides information for type of CVD. Scoring is accomplished by reading the color chip/ disc numbers on the reverse side and the sequence selected by the patient is recorded on a copy of the score sheet, for example 1,15,2,3,14,13,12,11,10,9,8,7,6,5,4. A patient with a CVD arranges the color discs in a different order than a person with normal CV. Subsequently, the diagramming is done and a circular results diagram is considered "pass" and an interlacing pattern is contemplated "fail/colour vision defective". The "interlacing pattern diagrams" are matched with standard diagrams on booklet depicting protanopia/ deuteranopia/tritanopia and the corresponding diagnosis or else "an undefined CVD" is inferred. Farnsworth Munsel D 15 test is relatively quick and easy to administer.

The Amsler's grid [original grid (with white lines on black background)] is used to examine central 20^0 of visual field i.e. area of retina providing fine details, at 1/3 m with opposite eye closed. The patient fixates on central spot with reading glasses "on" and reports blurry areas, scotoma, distorted lines or other defects of grid pattern. The test is done in each eye separately.

The contrast sensitivity (CS) was tested on Pelli Robson Charts (86 × 63 cm chart). It consists of 16 triplets of 4.9 cm (2.8° at 1 m) letters and assesses contrast sensitivity CS at a spatial frequency of about 0.50 to 1 cycle/degree. Within each triplet, the letters have the same contrast, and the contrast in each successive triplet decreases by a factor of 0.15 log units. The subjects read the chart at 3m and the result is recorded from "readings" given on the chart according to the last triplet which patient can see. The score ranges from 0.25 (least score) to 2.00 (best score).

The stereopsis is assessed by use of Titmus fly test. The polaroid vectograph is held at 40 cm ahead. The subject had to wear crossed polaroid filters (1 pair of standard 3D viewers) to present slightly different aspects of the same object to two eyes. The patient is passed thorough all three aspects of test that is –touching wings of housefly, seeing 9 sets of four circles and seeing three rows of five animals. The results for stereopsis test score is read from answer key and recorded.

The LV subjects having glaucoma have presentation similar to LV subjects with retinal diseases except for central vision preservation, more prominent impaired dark adaptation and defective night vision in glaucoma. These subjects present with diminished light sensitivity and they often pick up trouble when lighting is dim (e.g. cinema halls, dimly lit restaurants). Poor lighting makes seeing difficult. They have difficulty in distinguishing different shades of the same color i.e. contrast sensitivity is reduced. They also have glare and have trouble seeing in bright sunlight and at bright stores and extreme changes in lighting makes it hard for people with vision loss from glaucoma to see clearly.

Rehabilitation

The functional loss in LV is irreversible and there is a great need for low-vision rehabilitation. Most patients with LV are elderly and have functional limitations from other health problems that could add to the restraints caused by their visual impairments. Unattended low vision has far-reaching consequences: developmental effects in children, and functional, mental, socio-economic and quality of life implications for all those affected across the life spectrum. People with LV have residual vision with some light perception. Low vision aid (LVA) is a device that aims to make the most of the remaining vision. The individual's visual and environmental requirements are to be determined to find which types of orientation and mobility programs and devices are most effective

Magnification is the most widely used rehabilitation for LV subjects. The magnification devices stimulate more number of photoreceptors thus ensuring that signal necessarily goes to the brain for recognition of reading material or an object. Near magnification devices include hand held magnifiers (both illuminated and non-illuminated), stand magnifier, pocket magnifier, spectacle magnifier, etc. Distant magnification devices include telescopes and See TV glasses. With the help of closed circuit television, LV patients with reduced CS are able to enhance their residual vision to read scripts and notes by the manipulation of contrast, brightness, reverse polarity and magnification which are skills they acquire during their training in the use of the devices. The use of high contrast materials and reading text becomes helpful. The visual stimulation in young children through computer-based visual stimulation (VS) program combining checkerboard pattern reversal (passive stimulation) with oddball stimuli (attentional modulation) may be helpful.

To improve CS, LV subject needs letters to be made bold through use of bold chalks on chalkboard. If white board

is used, bold black markers are recommended over other colors. Reading text written with felt-tipped pens on paper and/or use of bold letters on tinted paper is of help. The corrective lenses with a yellow filter, preferably that which does not allow infrared rays to pass through, improves ability to discern contrast. The eye glasses with custom wave front lenses to reduce higher order aberration can also be utilized.

The glare can be reduced by using higher contrast settings when working with computers or electronic devices. There are also special modes, screens and hoods to help reduce glare. Other simple ways include covering surfaces that reflect light, closing curtains and changing sitting position so that less excess light is reflected from computer screen.

As there is no device to enhance stereopsis, the only suggested corrective measures is to improve VA with use of corrective lenses (to improve vision of affected eye or to block bad image), LASIK, eye rolling exercises, or resting the dominant/better eye.
Sectoral prisms can increase field of view allowing one to see more at once.

Low lights causes "dim" vision and having too little light reduces the ability of LV subject to see. Thus somewhat more than normal lights and utilising task lighting like desk and reading lamps and goose-neck lamps can help. When outside on cloudy and overcast days, LV subject can use amber or yellow lens glasses to help him see better.

Glare, caused by bright lights (sun or fluorescent lighting) is the excess light that comes into eyes from above or the sides of the visualised target. This extra light makes it harder to see and can be reduced by wearing hats outdoors or glasses with special lenses. Using sunglasses with tinted lenses (or contrast-enhancing filters generally yellow filter) can help reduce the effects of glare. A few different coloured lenses including brown, yellow or amber can be tried to find the ones that work best. But it is important to know that not all sunglasses are helpful. Lenses that are too dark can make it harder to see by dimming the light from front while letting in excess light from above and on the sides. Thus wrap-around glasses that block light coming in from above and the sides are the best for reducing glare e.g. in LV subjects with glaucoma.

For people with glaucoma-related vision loss, bigger does not necessarily mean better. People with tunnel vision often have trouble with high levels of magnification because of their lack of side vision and decreased field of view i.e. the amount of field one can see at a time. This type of vision loss does not benefit from magnifier devices that enlarge or make things bigger. Typically as magnification increases, the field of view decreases and the instability of the image (i.e. its shakiness) also increases. Thus too much magnification reduces peripheral field of vision even more and what is needed is the opposite of magnification, implying that there is a need for minimization (minification), or making the image smaller to increase LV subject's field of view (so that he can see more at one time). Minimization puts the peripheral images into the remaining central field of vision and provides more information in a smaller area by reducing or shrinking the intended target and fitting this into the remaining visual field enabling LV subject to see. Reverse telescopes (like holding binoculars backward) and high minus lenses minimize what one sees and help LV subject to see more within his reduced field of view.

People with glaucoma have a hard time adjusting their eyes to changes in lighting. Walking into a dark room or out of dark room into a bright area can temporarily stun the eyes so that they see less. This can be especially dangerous when walking or driving in areas that are light and dark (bridges, tunnels, shady areas). It can make it harder for LV subjects to see signs, steps, curves or changes in terrain. If one has glaucoma, he should take a few extra moments when there is a drastic change in light to let his eyes adjust.

Modern low vision rehabilitation (LVR), which took hold in the last few decades, has been propelled to new heights by relentless advances in basic and clinical sciences. Today we can provide significant and meaningful help to visually impaired patients in most situations. It could be as simple as a hand magnifier or as intricate as a retinal prosthesis. Devices and re-training of skills have been added to complement diagnosis and rehabilitation.[27]

Conclusions

Low vision rehabilitation (LVR) is today a recognized discipline in Ophthalmology, expanding and improving the quality of life of numerous visually impaired patients. It was not so about a century ago when charity work aimed at helping blind children was all that LVR was. With advances in science, medicine and public health policy, help for the blind expanded its reach to all who were visually impaired.[27] But LVR can only be achieved successfully provided we know how different aspects of vision change/deteriorate for a LV subject and what aspect is the most vital one to be improved upon. More studies on visual perception of LV patient in varied fields of visual functions and randomized controlled trials with intervention comparisons and outcome measures are needed to form stronger conclusions for the most effective low-vision rehabilitation interventions for individuals with LV.[21]

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