Research Journal of Medicine and Medical Sciences, 4(1): 85-88, 2009

Stereopsis and Contrast Sensitivity Binocular Summation in Early Glaucoma

[1]Amal A. El-Gohary and [2]Ghada A. Siam

[1]Ocular Physiology and [2]Ophthalmology Departments, Research Institute of Ophthalmology, Giza, Egypt.

Abstract: Purpose: To investigate the stereoacuity and binocular summation for contrast sensitivity in early glaucoma patients. Methods: The study included 18 patients with early glaucoma and 15 controls. Both groups were evaluated for depth perception using Lang stereotest and binocular summation using VCTS contrast sensitivity chart at 1.5, 3, 6, 12, and 18 spatial frequencies. Results: Stereoacuity was significantly reduced in early glaucoma patients. Binocular contrast sensitivity function (CSF) was significantly reduced across spatial frequency range from low spatial frequency (1.5c/d) to high spatial frequency (18c/d). Binocular summation ratio(BSR) was decreased below normal value at spatial frequencies 1.5, 3, 18 c/d. The robust decrease of BSR was observed at spatial frequency 3 c/d. Conclusions: We concluded that in early glaucoma the binocular mechanisms that mediate binocular summation and stereoacuity are disrupted. VCTS contrast sensitivity chart and Lang-stereotest could be sensitive measures for testing visual function in early glaucoma patients.

Key words: Early glaucoma, stereopsis, contrast sensitivity and binocular summation.

INTRODUCTION

The disability caused by glaucoma is related pathologically to the progressive damage of the retinal ganglion cells[23] such damage is manifested structurally by the appearance of the optic disc and functionally by the characteristic changes in the visual field. However, Glaucoma is not only a major neurodegenerative disease of visual neurons in the eye, but also in the brain[27,21]. Therefore, detecting the functional damage can be more thoroughly done by evaluating the binocular visual functions such as stereopsis and binocular contrast sensitivity[3,7].

Stereopsis is thought to reflect the disparity tuning characteristics of cortical neurons. However, the ability to detect visual stimuli is also better for binocular versus monocular vision, a phenomenon referred to as binocular summation[2,1]. Stereopsis and binocular summation are indication of binocular advantage. Although binocular cortical neurons are likely the substrate for both stereopsis and binocular summation[11] several reports suggest that different binocular units probably mediate these two different abilities[4,12].

In early glaucoma patients instances of visual dysfunction has been demonstrated for spatial, temporal and motion thresholds. Although these visual abilities are typically disrupted in glaucoma, it is not clear whether neuronal mechanisms that binocularly combined this monocular input (neural summation) are themselves disrupted. Previous investigation of binocular summation of contrast sensitivity in glaucoma was carried on by using letter chart[7]. That measure contrast sensitivity at only one spatial frequency. On the other hand, if binocular summation is measured at a wide range of spatial frequencies it will provide a more comprehensive evaluation.

In the present study we were aiming to investigate the stereoacuity and binocular summation for contrast sensitivity in early glaucoma patients.

MATERIALS AND METHODS

The study included 18 patients with early glaucoma and 15 controls. Age ranged in the patient group from 45 to 68 years, and for the controls from 46 to 58 years. The diagnosis of early glaucoma was established by the appearance of the optic nerve and visual field loss detected by perimeter. Disc Damage Likelihood Scale (DDLS) was used to evaluate the optic nerve. Visual field tests were conducted using Humphrey Field Analyzer performing a central 24-2 threshold test procedure; then the Glaucoma Staging System 2 (GSS2) was used to classify the field results. All the included patients were classified as having early loss. Patients and controls had visual acuity of 0.7 or better. Complete clinical ocular examination was done to all subjects; including anterior segment examination with the slit-lamp biomicroscopy, applanation tonometry and gonioscopy and fundus examination with indirect ophthalmoscope through dilated pupil.

Corresponding Author: Amal A. El-Gohary, Ocular Physiology Department, Research Institute of Ophthalmology, Cairo, Egypt.
E. mail: a-gohary@hotmail.com

Stereoacuity: The stereoacuity was measured with the Lang-Stereo test; a random-dot stereo test uses a panographic technique to present disparity, Subjects were tested under the same condition ; and they had no previous experience with this type of test no glasses were required. The test cards were viewed at a distance of 40 cm and the patient was asked if he or she can see a picture of a cat in the card, if the answer was yes, then he or she was asked to point to its site, if the subject said no, then he or she was allowed to change the visual axes of the card to detect the picture .This was repeated with all disparities starting with Lang I then Lang II. The lowest disparity which the patient can reliably discriminate was recorded and this stereothreshold was the measure of stereoacuity.

Contrast Sensitivity: For each subject contrast sensitivity function (CSF) was determined using the VCTS 6500 chart: The chart is viewed at a distance of 3.05 m at a luminance level of 100 cd/m^2. The spatial frequencies tested were ranging between 1.5 and 18c/d (cycle per degree). The observers were wearing their best distance correction. Each eye was tested separately, then binocularly to determine the monocular and binocular CSF. The contrast of the correctly identified target in each row is scored as the contrast threshold for that spatial frequency. Binocular summation ratio (BSR) for each spatial frequency was calculated as the quotient of the binocular CSF divided by the best eye monocular CSF (Rager *et al.*, 2003). The true values of contrast at each spatial frequency were used.

RESULTS AND DISCUSSION

Results: The mean age of control subjects was (53.26±4.14) and that of early glaucoma patients was (56.00±6.30). No statistical difference was found between the two groups (p=0.164)

Stereoacuity Results: The mean stereothreshold of the age matched control subjects was (200) and that of early glaucoma patients was (494.44±88.92). The mean stereothreshold of early glaucoma patients was significantly higher compared to control subjects(p=0.000) Fig. (1).

Contrast Sensitivity Results: The mean values of binocular CSF of early glaucoma patients and best monocular performance and BSR were summarized in Table (1). The binocular CSF of glaucoma patients were significantly low at all spatial frequency compared to control subjects p =0.019, at 1, 5 c/d, p= 0.000 at 3, 6, 12 c/d and p= 0.002 at 18 c/d. Except the 1.5 c/d (p=0.881), the best eye CSF of glaucoma patients were significantly low compared to control subjects p =0.000 at 3, 12 c/d, p=0.001 at 6 c/d and p=0.006 at 18 c/d. (Table 1and Fig. 2). Binocular summation ratios for the control subjects and patients with early glaucoma are shown in Fig. (3) and Table (1). All controls showed normal binocular summation ratios that were consistent with neural binocular summation (~1.4 or above). The BSR of early glaucoma patients were close to unity (1.2) at 3, c/d gratings. There was significant difference in comparison with control subject value (p=0.015). The BSR values at 1.5, 6 and 18c/d were ~1.3. Although these were above unity but still blow the normal values. In comparison to control subjects significant difference was found at 18 c/d (p=0.016). BSR increased to normal value (1.45) at 12 c/d. but it was significantly low in comparison to controls (p=0.022).

Discussion: Glaucoma is a neurodegenerative disease of the visual system. So the identification of patterns of neuronal death and injury is an intense area of investigation requiring reliable methods to assess the effects of neuronal disruption on visual performance which can lead to early detection of glaucoma[25]. This study showed that in early glaucoma patients' binocular and most monocular CSF for stationary grating stimuli were significantly reduced across spatial frequency range from low spatial frequency (1.5c/d) to high spatial frequency (18c/d). These findings indicate that low, med and high spatial frequency sensitive channels were approximately equivalently impaired in early glaucoma. Previous studies reported that binocular contrast sensitivity was affected very early in the disease[3,7]. Furthermore, we used Lang-stereotest and the results showed that steroacuity was significantly reduced in early glaucoma patients. This is in agreement with other studies using different stereotests and confirms the sensitivity of Lang-stereotest[3,10]. Stereoacuity has been suggested to be mediated through M pathway Livingstone & Hubel,[14]. For static pattern the M pathway has not been demonstrated to play any role in detecting threshold pattern at high med or even low spatial frequencies. These patterns are mediated by the P pathway[19,18,17]. Further, binocular contrast sensitivity as well as stereoacuity was significantly reduced in early glaucoma patients. These results suggested that both parvocellur (P) and magnocellur (M) pathways are disruption early in the disease. Our findings are in agreement with McKendrick *et al.*[15]. Recent laboratory research revealed that selective large (M) cell loss may be more complicated than initially thought. Both large and small (P) cells shrink before cell death in experimental glaucoma, but those large cells may be more susceptible to this process[20,26]. This binocular neural disruption may be at the level of

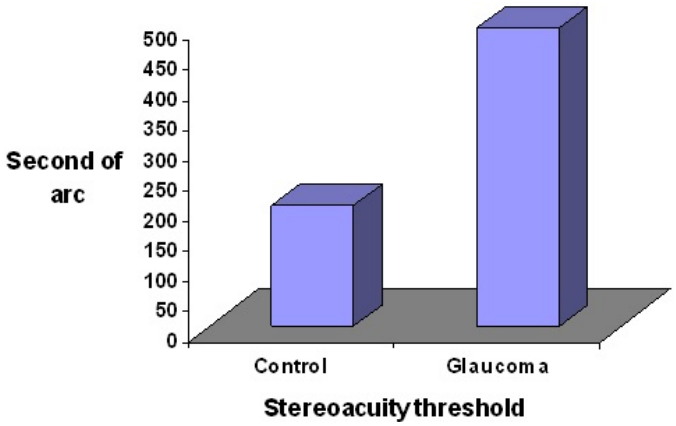

Fig. 1: Mean stereothreshold in seconds of arc for control subjects and glaucoma patients.

Pro-inflammatory and Anti-inflammatory Cytokines in Vitreous Fluid of Patients with Proliferative Diabetic Retinopathy

Pro-inflammatory and Anti-inflammatory Cytokines in Vitreous Fluid of Patients with Proliferative Diabetic Retinopathy

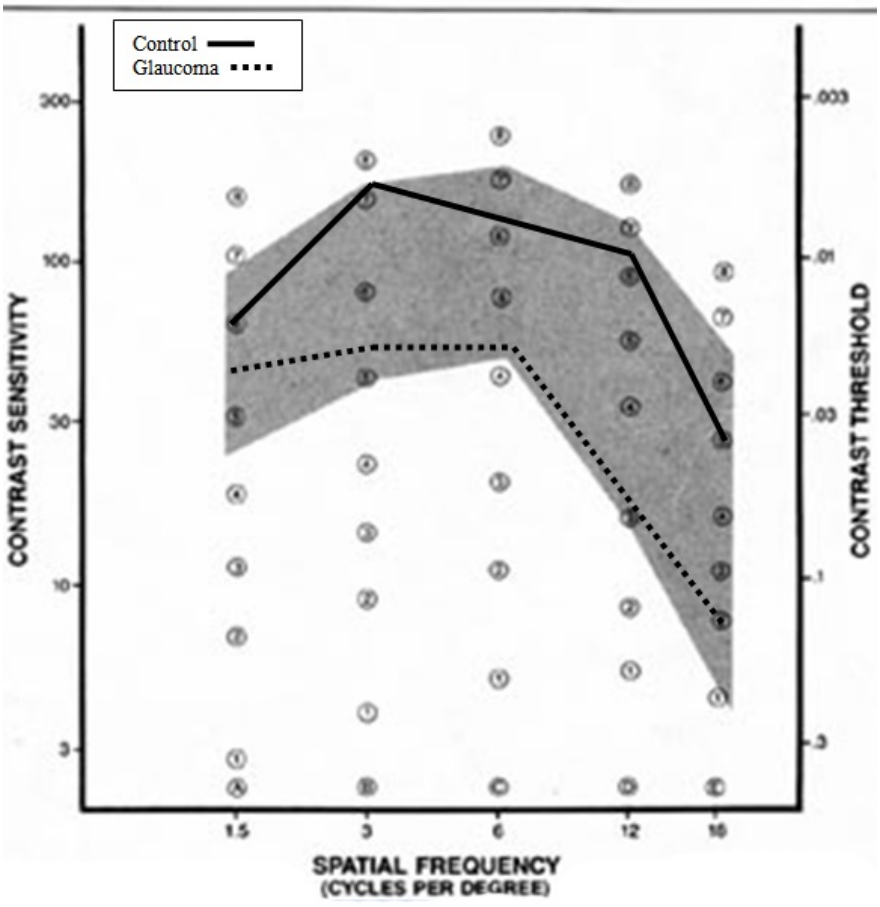

Fig. 2: Binocular contrast sensitivity curves for control subjects and glaucoma patients.

Table 1: The mean values± SD of binocular CSF, best eye CSF and BSR of glaucoma patients and controls (according to VCTS chart).

Parameters	Control subjects(15)			Glaucoma patients (18)		
Spatial Frequency	Best eye CSF	Binocular CSF	BSR	Best eye CSF	Binocular CSF	BSR
1.5 c/d	50.00±17.32	70.00	1.56±.49	46.66±16.97	58.33±16.97*	1.33±.48
3 c/d	90.66±21.94	170.00±38.90	1.66±.48	55.44±22.39*	62.22±20.96*	1.19 ±.37*
6 c/d	99.66±36.02	138.00±44.83	1.42±.33	44.66±33.63*	52.77±31.70*	1.31±.48
12 c/d	59.40±26.67	94.40±27.32	1.77±.55	11.88±8.02*	17.66±14.76*	1.45±.43*
18 c/d	18.06±14.54	29.33±23.32	1.68.±53	5.83±2.54*	7.38±3.63*	1.30±.51*

*significant p< 0.05.

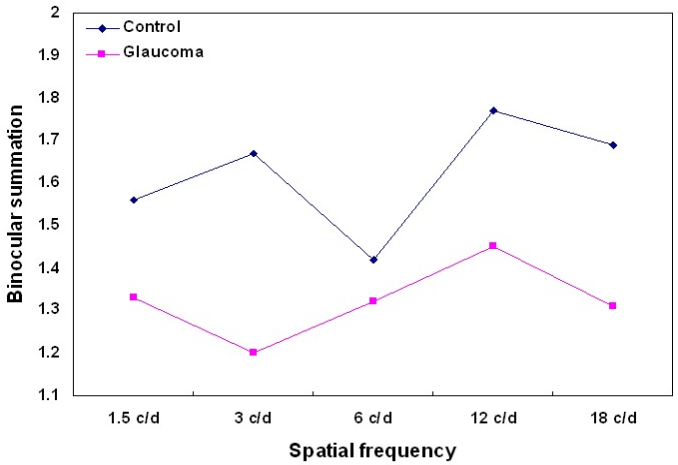

Fig. 3: Binocular summation ratio (BSR) curves for control subjects and glaucoma patients.

retinal ganglion cells or extend to higher levels; the geniculo-cortical pathways and cortical neurons as evidenced in experimental [13] and human[10] studies. In addition to Disparity-tuned binocular neurons which are thought to mediate stereoacuity, other flat binocular neurons show a summation of monocular stimulation in a fashion not selective for depth[22].

In most patients with glaucoma there is a considerable asymmetry between the two eyes as regards to the amount of functional and pathological damage .this may be manifested as impairment in binocular vision. The binocular summation ratio is the ratio of binocular to monocular sensitivities, often measured for sine-wave gratings[6]. For example, a binocular summation ratio of unity indicates no binocular advantage, whereas a ratio of two indicates that contrast sensitivity is twice as high with two eyes as with one. The binocular summation ratio for normal observers is ~1.4 (√2), or, typically, slightly higher[8,1,16] and is attributed to physiological summation of the two monocular signals[1].

The obtained results of this study showed that BSR for contrast sensitivity (by using VCTS grating chart) were normal in control subjects at all spatial frequencies tested. In glaucoma patients BSR was decreased below normal value at spatial frequencies 1.5, 3, 18 c/d .The robust decrease of BSR was observed at spatial frequency 3 c/d that reached unity (1.2) suggesting an increase in the interocular sensitivity difference as well as a decline in the neural integration of binocular contrast information. These results disagree with Essoch and his associates. They did not find significant decrease in BSR (~1.7) in early glaucoma patient[7].

The difference could be explained by the different charts used. Pelli-Robson chart is a letter chart designed to measure contrast sensitivity at ~3c/d while VCTS chart is a grating chart that measure contrast sensitivity at a wide range of spatial frequencies which is more suitable for measuring BSR. The neural basis for binocular summation has been suggested to be an increase in the sensitivity of the cells under binocular as compared to monocular stimulation. That is rather than additional units being play under binocular condition, most cells become more sensitive, and the most sensitive cells determine the threshold of binocular summation[7]. We concluded that in early glaucoma the binocular mechanisms that mediate binocular summation and stereoacuity are disrupted. VCTS contrast sensitivity chart and Lang-stereotest could be sensitive measures for testing visual function in early glaucoma patients and simple techniques may help in early diagnosis of glaucoma.

REFERENCES

1. Baker, D.H., T.S. Meese and M.A. Georgeson, 2007. Binocular interaction: contrast matching and contrast discrimination are predicted by the same model. Spat Vision, 20: 397–413.

2. Baker, D.H., T.S. Meese and R.J. Summersm, 2007. Psychophysical evidence for two routes to suppression before binocular summation of signals in human vision. Neuroscience, 146: 435–448.
3. Bassi, C.J. and JC. Galanis, 1991. Binocular visual impairment in glaucoma. Ophthalmology, 98: 1406-1411.
4. Blake, R. and R. Fox, 1973. The psychophysical inquiry into binocular summation. Percep. Psychophys, 14: 161-185.
5. Blake, R., M. Sloane and R. Fox, 1981. Further developments in binocular summation. Percep Psychophysics, 30: 266–276.
6. Campbell, F.W. and DG. Green, 1965. Monocular versus binocular visual acuity. Nature, 208: 191–192.
7. Essock, E.A., R.D. Fechtner, T.J. Zimmerman, W.J. Krebs and J.D. Nussdorf, 1996. Binocular functions in early glaucoma. J. Glaucoma, 5: 395-405.
8. Georgeson, M.A. and T.S. Meese. 2005. Binocular summation at contrast threshold: a new look. Perception, 34(suppl): 141.
9. Gupta, N., L.C. Ang and Noel de Tilly *et al*., 2006. Human glaucoma and neural degeneration in the intracranial optic nerve, lateral geniculate nucleus and visual cortex of the brain. Br. J. Ophthalmol., 90: 674-678.
10. Gupta, N., N. Krinshnadeve, S.J. Hamstra and Y.H. Yucel, 2006. Depth perception deficits in glaucoma suspects. Br. J. Ophthalmol., 90: 979-981.
11. Hubel, D.H. and T.N. Wiesel. 1962. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. J. Physiology, 160: 106-154.
12. Hubel, D.H. and M.S. Livingstone, 1990. Color and contrast sensitivity in the lateral geniculate body and primary visual cortex of the macaque monkey. J. Neurosci., 10: 2223-37.
13. Lam, D.Y., P.L. Kaufman and B.T. Gabelt *et al*., 2003. Neurochemical correlates of cortical plasticity after unilateral elevated intraocular pressure in primate model of glaucoma. Invest. Ophthalmol. Vis. Sci., 44: 2573-2581.
14. Livingstone, M.S. and D.H. Hubel, 1987. Psychophysical evidence for separate channels for the perception of form, color, movement, and depth. J. Neuroscience, 7: 3416-3468.
15. McKendrick, A.M., G.P. Sampson, M.J. Walland and D.R. Badcock, 2007. Contrast sensitivity changes due to glaucoma and normal aging: low spatial frequency losses in both magnocellular and parvocellular pathways. Invest. Ophthalmol. Vis. Sci., 48(5): 2115-2122.
16. Meese, T.S., M.A. Georgeson and D.H. Baker, 2006. Binocular contrast vision at and above threshold. J. Vis., 6: 1224–1243.
17. Merigan, W.H. and T.A. Eskin, 1986. Spatiotemporal vision of macaques with severe loss of P-beta retinal ganglion cells. Vision Res., 26: 1751-61.
18. Merigan, W.H. and H.R. Maunsell, 1990. Macaques vision after magnocellular lateral .eniculate lesions. Visual Neurosci., 5: 347-52.
19. Merigan, W.H. and M. Pand, 1992. Specialization in the macaques. Visual Neurosci., 8: 575-83.
20. Morgan, J.E., 2002. Retinal ganglion cell shrinkage in glaucoma. J. Glaucoma, 11: 365-370.
21. Nucci, C., M. Bari, A. Spanò, M. Corasaniti, G. Bagetta, M. Maccarrone and LA. Morrone, 2008. Potential roles of (endo) cannabinoids in the treatment of glaucoma: from intraocular pressure control to neuroprotection. Prog. Brain. Res., 173: 451-64.
22. Poggio, G.F., 1995. Stereoscopic processing in monkey visual cortex: a review. In Papathomas T.V., Chubb C., Gorea A., kawler E. Eds. Early vision and beyond. Cambridge, M.A: MIT Press, 43-53.
23. Quigley, H.A., G.R. Dunkelberger and W.R. Green, 1988. Chronic human glaucoma causing selectively greater loss of large optic nerve fibers. Ophthalmology, 95: 357-363.
24. Rager, W.C. R.W. Gagnon and D.W. Kline, 2003. Senescent effects on binocular summation for contrast sensitivity and spatial interval acuity. Curr. Eye Res., 27(5): 315-21.
25. Varma, R., P. Peeples, J.G. Walt and T.J. Bramley, 2008. Disease progression and the need for neuroprotection in glaucoma management. Am. J. Manag. Care, 14(1 Suppl): S15-9.
26. Weber, A.J., P.L. Kaufman and W.C. Hubbard, 1998. Morphology of single ganglion cell in the glaucomatous primate retina. Invest. Ophthalmol. Vis. Sci., 39: 2304-2320.
27. Yücel, Y. and N. Gupta, 2008. Glaucoma of the brain: a disease model for the study of transsynaptic neural degeneration. Prog. Brain. Res., 173: 465-78.