January - February 2020
ISSN: 0193 - 4120 Page No. 11845 - 11851

Saccadic Suppression in the Magnocellular Pathway during Reading

Parameswari Shunmugam1*, Vijayakumar2, Nachiappan Murugavadigal3,
Kalai Arasu Muthusamy1
1Faculty of Medicine, Universiti Malaya, Kuala Lumpur, Malaysia
2Faculty of Computing and Informatics, Multimedia University, Cyberjaya, Malaysia
3Department of Surgery, Melaka Manipal Medical College, 75150 Melaka, Malaysia

Article Info
Volume 82
Page Number: 11845 - 11851
Publication Issue:
January-February 2020

Article History
***Article Received**: 18 May 2019*
***Revised**: 14 July 2019*
***Accepted**: 22 December 2019*
***Publication**: 21 February 2020*

Abstract

Abstract: Movement detection and acuity resolution deferentially affect the magnocellular, parvocellular and koniocellular pathways of the human visual neural system. Temporal and spatial frequency sensitivity measurements are used to identify the neural behaviour of these pathways. The magnocellular pathway (MP) is known to carry a limited range of temporal frequency components of signals resulting from a visual scene. In this paper, we show a possible mechanism which will give rise to a fixed bandwidth of the temporal frequencies which can be conveyed by the MP during fixations and saccades. We will also explain why temporal frequency components outside this bandwidth are not available for the MP. We have proposed two factors to explain our results. The first, which is neural in origin, is the correlated firing mechanism of the parasol ganglions which serve the MP and the second, which is biophysical in origin, is the windowing effect of saccades and fixations. Using the two mechanisms, we will show that the allowed temporal frequencies in the MP have an unique upper and a lower bound. The upper bound arises from the fixed correlated firing time of the parasol retinal ganglions. The lower bound is calculated using the windowing effect of eye fixations and saccades. We will show how the combined effect of the correlated firing the retinal ganglions and the windowing effect of fixations and saccades provides an explanation for the various aspects of the observed phenomenon known as saccadic suppression.

Keywords: *EEG, Magnocellular, Reading, Saccade, Saccadic Suppression, Dyslexia*

I. INTRODUCTION

The visual neural system includes two major parallel streams, the magno-cellular and parvocellular pathways (MP and PP). The magnocellular pathway (also known as the transient system) obtains its input from the parasol ganglions in the retina of the eye and responds to rapid changes in visual scenes. It carries signals from the peripheral region outside the fovea, thus contributing to peripheral vision. The visual signals are then sent to the lateral geniculate nuclei (LGN) of the thalamus and then projected via the visual cortex and the dorsal or "where" stream to the parietal cortex. [1]- [4].

A. Saccadic Suppression

Saccades are rapid eye movements that take place in the eye and they occur about three times a second, each lasting about 25 ms each. During reading, these saccades take place in between longer periods of eye fixation which usually last about 250 ms each [1]. When the eye moves rapidly, motion of the stationary world is generally not perceived despite displacement of the whole image on the retina. This reduction of visual sensitivity around the time of a saccade is known as saccadic suppression. While perisaccadic suppression describes suppression of visual information around the time of a saccade, it has been found that this suppression is present even long before the onset of a saccade and also long after the saccade, known as pre-saccadic and post-saccadic suppression respectively[5]. Various mechanisms, both active as well as passive have been proposed as candidates for saccadic suppression [6]-[7]. The nature and modification of the neural signals in the MP is particularly relevant in studies relating to learning disorders. The Magnocellular Theory of Developmental Dyslexia, as proposed by John Stein advocates a visual magnocellular deficit among dyslexics [8].

B. Correlated Firing of Retinal Ganglions

The correlated firing time of the parasol retinal ganglion cells fall within a fixed time regime [9, 10]. We will show in this paper that the fixed correlated firing time imposes an upper limit on the temporal frequencies of the signals that can be conveyed by the MP. The correlated firing times from human retinal ganglions are not available [9]-[10], hence data from flicker studies in animals [1,11] have been used. Correlated firing refers to the firing of groups of similar cell types such as the parasol ganglions which take place in a highly correlated and simultaneous manner. Different cell types have specific timing regimes. The correlated activity is seen in diverse neuronal systems such as the the retina, cochlea, spinal cord, cerebellum, hippocampus, and neocortex and is observed in several species throughout their development. This suggests that it is an essential phenomenon in neural circuitry. Understanding the role of correlated neural activity has been an active area of study for several years [9, 10, 12, 13].In this paper we examine how correlated firing of the parasol ganglions play a role in determining the upper limit of the temporal frequency sensitivity. Since it is known that each cell type has its own timing regime for correlated activity [14], the magnocellular pathway will have an unique upper limit in the temporal frequency sensitivity. These upper limit can be experimentally measured by contrast sensitivity and flicker rate studies[15, 16, 17].Animal retinal neurons has been known to fire synchronously within three ranges, i.e., 40 to 100 ms (broad), 10 to 50 ms (medium), and below 1 ms(narrow) [9, 14]. It is also known that the synchrony occurs according to each cell type i.e. Parasol, midget, and small bistratified cells firing together respectively to convey visual signals to the magnocellular, parvocellular and koniocellular pathways respectively. Using data from animal studies [9],[10],[14],[18],[19], we have assumed a correlated firing time of 10 ms for the parasol cells for the purpose of illustration. Correlated firing after 10 ms implies that this group of ganglion cells will wait up to 10 ms then fire together

II. METHODOLOGY AND RESULTS

In this section we show how the allowed upper and lower bounds of the temporal frequencies in the magnocellular pathway are found. We have used the correlated firing mechanism of the parasol ganglions as well as the windowing effect of fixations and saccades to calculate the temporal frequency bounds. We have then used sample electroencephalographic (EEG) data to identify regions of the scalp which show saccadic suppression.

Calculation of upper bound of signal frequency

If correlated firing in parasol ganglions is 10 ms, these ganglions sample the available visual signal and fire simultaneously after 10 ms. The Nyquist criterion limits the highest possible frequency that can be detected, hence, the sampling rate according to the Nyquist criterion, should be at least twice the highest available frequency. In flickering visual signals, flicker frequency rates that will be detected must be below half of $1/10\times10^{-3}$ Hz = 100 Hz, i.e. 50 Hz. Flicker rates higher than this will imply require ganglion correlated firing times less than 10 ms. Such signals i.e temporal frequencies above 50 Hz will not be detected since the midget ganglions are still waiting to fire.[1]

A. Calculation of lower bound of signal frequency.

The lower bound of the temporal frequencies of the signals conveyed by the MP is obtained by the windowing effect imposed on the time domain signals before they are seen by the retinal ganglions.

Two windowing possibilities are available in the human visual system, i.e. windowing effect due to fixation and another windowing effect due to saccades. Fixation has a window length of about 250 ms and eye saccades have a window length of about 25 ms[1, 20]. A perfect rectangular time window of a length of T seconds, imposed on any signal gives rise to a lower bound of the frequencies that can be transmitted i.e f= 1/T Hz. For non-rectangular windows the lower bound of the frequencies reduces to ½ f Hz [21]. Due to the finite time required for the beginning and ending of fixations and saccades, we assume a non-rectangular window for both cases. Assuming that the correlated firing time,t_c, for the parasol ganglions is 10 ms and and the eye fixation time,T_f is of length 250 ms, we note that the eye fixation time is higher than the correlated firing time of the parasol cells. Hence, visual signals will enter the MP without problems. Note that parasol ganglions will fire about 250/10 = 25 times during the saccade giving rise to a strong magnocellular signal. The lower bound of the temporal frequency is $½ (1/T_f) = 2$ Hz. This implies that temporal signal changes below 2 Hz cannot be detected by the MP during fixation. Figure 1 is an illustration of typical neural excitations traversing the MP during fixation. During the progression of an eye saccade, the saccade time, T_s is 25 ms which is larger than the correlated firing time of the parasol ganglions, visual signals will thus enter the MP during saccades. Note that parasol ganglions will only fire about 25/10 = 2.5 times during the saccade which will give a much reduced signal compared to that during fixations. Further, during saccades, the lower bound of the temporal frequency of $½ (1/T_s) = 20$ Hz. This implies that temporal signal changes below 20 Hz cannot be detected by the MP during saccades. Figure 2 is an illustration of typical neural excitations traversing the MP during a saccade. The results are tabulated in Table 1.

Table 1: Upper and lower bounds for signal frequencies in the MP during fixations and saccade

Window	Width (ms)	Upper Bound (Hz)	Lower Bound (Hz)
Fixation	250	50	2
saccade	25	50	20

During fixations, signals with temporal frequencies that the MP will be able to detect lie between 2 Hz and 50 Hz. During saccades the MP detects temporal frequencies between 20 Hz and 50 Hz. Results by numerous authors substantiate this finding [22], [15], [16], [17].

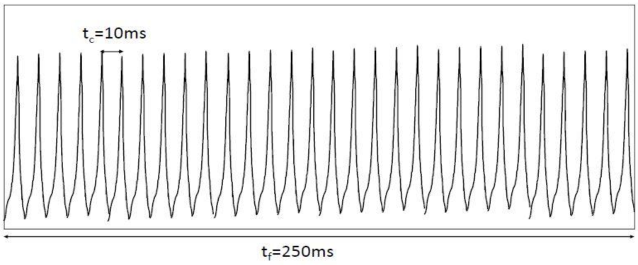

Figure 1: Simulated neural signals in the MP based on thefiring of parasol ganglions every 10 ms during a fixation window of 250 ms. Fixation time (t_f) = 250 ms, Correlated firing time of parasol ganglions (t_c) = 10 ms.

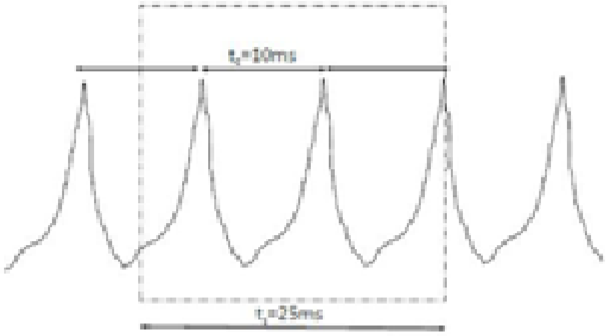

Figure 2: Simulated neural signals in the MP based on the firing of parasol ganglions every10 ms during a saccade window of 25 ms. Saccade time(t_s) = 25 ms, Correlated firing time of parasol ganglions (t_c) = 10 ms.

B. Saccadic Suppression from EEG data

We have used sample electroencephalographic data to identify saccadic suppression at various electrode locations on the scalp. The sample dataset was obtained from the EYE-EEG toolbox website [21], [22]. The data is obtained for a natural reading experiment.

Participant read lists of five words from left to right.There were 320 epochs and each epoch represents a single word that is read. For each epoch a period of silence of 1 second (from -1000 to 0 ms) preceded the reading stimulus which lasted 2 seconds (from 0 to 2000 ms). Binocular eye movements were recorded with an Eyelink 1000 tracker at 1000 Hz. A 72 channel EEG data was recorded with Biosemi Active amplifiers at a rate of 512 Hz. The EEG data was band-pass filtered from 0.1 to 100 Hz and converted to average reference. Online detection of saccades, fixations and blinks was switched on. Eye movement events can be directly imported using EYE-EEG. EEGLAB[23],[24] running in Matlab was used together with the EYE-EEG toolbox to identify saccades and fixations.

EYE-EEG toolbox was used to clean the data. Whole epochs are discarded that contain any out-of-range eye tracking data. The time-frequency toolbox in EEGLAB was used to obtain event-related spectral perturbation (ERSP) plots. The ERSP plots, show EEG frequencies versus time with colors used to identify intensity.

During saccades it is believed that neural signals are suppressed and as such, noise dominates. The rapid oscillations of noise gives rise to high frequency components known as gamma bands (>30Hz) in EEG spectra. Figure 3 shows (in red) the time of onset and progression of statistically significant ($p<0.05$) gamma bands at various scalp locations after stimulus 270ms (AF7), 350ms (AF8), 400ms (C5), 350ms (C4), 350ms (O1) and 350ms (O2) based on the 320 individual reading data epochs.

Figure 3 shows, gamma bands (shown as dark red, >30Hz) are used as markers for saccade events[25],[26].At the left frontal electrode (AF7), a saccade was registered at 300ms, and suppression (in dark blue) seen at 8-30 Hz and 300 to 500 ms. At the right occipital electrode O2, suppression follows the saccade registered at around 350 ms, from 70 – 90 Hz and 350 to 450 ms.

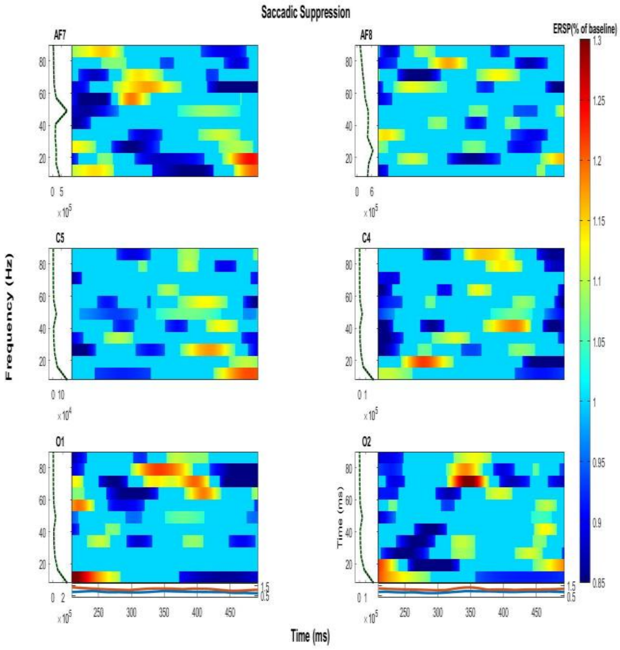

Figure 3: Saccades are indicated by gamma bands (in red) at around 300 – 350 ms and above 30 Hz. Suppression is shown in dark blue in electrodes AF7: <40Hz, O2, >70Hz.

III. DISCUSSION AND CONCLUSION

The results were obtained using known correlated firing data from primates and it is assumed that they fall in the order of the range in humans. Intrasaccadic contrast sensitivity changes could be caused by neural optical, biophysical, or mechanical factors[29]. We have proposed two factors, the first is neural in origin, the correllated firing of parasol ganglions and the second is biophysical, which is the windowing effect of saccades and fixations. During fixations lasting about 250 ms, the temporal frequencies detected by the MP are band limited within 2 Hz and 50 Hz. During saccades lasting about 25 ms, the temporal frequencies detected by the MP are band limited within 20 Hz and 50 Hz. The correlated firing mechanism is like a sampling device. It samples a visual scene regularly to obtain information. In this case, when the temporal changes in a scene to be sampled is shorter than the sampling time itself, then it will be unable to extract any information about the temporal changes in the scene. The upper limits of the frequencies were found using straight forward signal analysis methods. Other researchers have indicated high frequency gamma oscillation 30 Hz in the MP and low frequency theta oscillations in the PP[1, 11, 30, 31, 32, 33]. Our analysis indicate that within the scope of the correlated firing data, the magnocellular pathway can detect temporal frequency changes up to 50 Hz.

It is also interesting to note that there is a close link between dyslexics and eye saccades which have been suggested by numerous authors [8, 34]. Note that the increase in the window size of eye saccades i.e. longer saccades, will bring down the lower bound of signals entering the magnocellular pathway during saccades.

A. Magnocellular signals during Fixation and Saccades while reading.

The correlated firing time of the parasol ganglions is 10 ms i.e. also lower than the fixation time of 250 ms. Thus the sufficiently long fixation time allows the ganglions to sample the visual scene every 10 ms during the fixation time. Hence, during fixations, the MP receive visual inputs. The correlated firing time of the magnocellular ganglions of 10 ms is also lower than the saccade time. Thus the time window of a saccade (25ms) allows the parasol ganglions to sample the visual scene every 10 ms during the progression of a saccade. saccadic motion. This indicates that the MP will be able to detect signals with temporal frequencies from 20 Hz to a maximum of 50 Hz. Note that during saccades, visual signals with temporal frequencies below 20 Hz will not be distinguishable from a stationary signal(DC)[35],[27].

B. The Magnocellular Deficit Theory

The allowed temporal frequencies of the visual signals while reading in the MP during periods of fixations and saccadic activity have been calculated. In reading, during the 250 ms period of a fixation the parasol cells serving the MP can fire up to 25 times since they have a correlated firing time of10 ms. This phenomenon will explain the large sensitivity in the MP during fixations. During saccades, we can see that although the magnocellular is activated[32] the parasol cells serving the MP can fire at least 2 times since they have a correlated firing time of 10 ms. This will reduce the magnocellular signal when compared to its activity during fixations. This can explain the phenomenon of saccadic suppression in the MP as discussed by several authors[2],[37],[38]. Note that larger saccade time intervals will increase the number of firing ganglions that are detected and smaller saccade time intervals will reduce the number of firing ganglions detected. The result from this study also indicates that visual sensitivity tests require flicker frequencies beginning from 2 Hz up to at least 50 Hz to comprehend the nature of saccadic suppression better During fixations as shown in Table 2, one would expect good signal amplitude in the MP from 2 Hz up to 50 Hz. During this period, we can expect the parasol ganglions up to 25 times. During saccades as shown in Table 2, there will be no signals in the MP at flicker frequencies below 20 Hz. From 20 to 50 Hz the MP responds to flicker. The parasol ganglions can fire only around 2.5 times resulting in reduced magnitude of the neural signals. Suppose the saccade window reduces from 25 ms to 15 ms, then the parasol ganglions can only fire only once per saccade, further reducing the magnitude of the magnocellular signal.

IV. CONCLUSION

In conclusion, the combined effect of correlated firing of the parasol ganglions and the windowing effect of fixations and saccades provides a possible explanation for the observed phenomenon known as saccadic suppression [2], [8],[33],[34]. Using EEG, saccadic suppression has been recorded by various electrodes on the scalp. Depending on electrode location, suppression has been observed before, during and after saccade events. Note that this observed changes in the neural signals quality in the magnocellular pathway has been understood without any active or passive suppression by any other agent.

V. REFERENCES

[1] K. Pammer, Temporal sampling in vision and the implications for dyslexia, Frontiers in human neuroscience 7 (2013).

[2] J. Stein, J. Talcott, Impaired neuronal timing in developmental dyslexia—the magnocellular hypothesis, Dyslexia 5 (1999) 59.

[3] B. C. Skottun, The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity, Vision research 40 (2000) 111–127.

[4] S. Kristensen, F. Garcea, B. Mahon, J. Almeida, Temporal frequency tuning reveals interactions between the dorsal and ventral visual streams, J Cognitive Neurosci 28 (2016) 1–8

[5] F. Bremmer, M. Kubischik, K.-P. Hoffmann, B. Krekelberg, Neural dynamics of saccadic suppression, Journal of Neuroscience 29 (2009) 12374–12383.

[6] A. Frost, M. Niemeier, Suppression and reversal of motion perception around the time of the saccade, Frontiers in systems neuroscience 9 (2015) 143.

[7] E. Castet, G. S. Masson, Motion perception during saccadic eye movements, Nature neuroscience 3 (2000) 177.

[8] J. Stein, The magnocellular theory of developmental dyslexia, Dyslexia 7 (2001) 12–36.

[9] I. H. Brivanlou, D. K. Warland, M. Meister, Mechanisms of concerted firing among retinal ganglion cells, Neuron 20 (1998) 527–539.

[10] M. Greschner, J. Shlens, C. Bakolitsa, G. D. Field, J. L. Gauthier, L. H.Jepson, A. Sher, A. M. Litke, E. Chichilnisky, Correlated firing among major ganglion cell types in primate retina, The Journal of Physiology 589 (2011) 75–86.

[11] T. N. Wiesel, D. H. Hubel, Spatial and chromatic interactions in the lateral geniculate body of the rhesus monkey., Journal of Neurophysiology 29 (1966) 1115–1156.

[12] A. G. Blankenship, M. B. Feller, Mechanisms underlying spontaneous patterned activity in developing neural circuits, Nature Reviews Neuroscience 11 (2009) 18–29.

[13] N. Dehorter, L. Vinay, C. Hammond, Y. Ben-Ari, Timing of developmental sequences in different brain structures: physiological and pathological implications, European Journal of Neuroscience 35 (2012) 1846–1856.

[14] M. Meister, M. Berry, The neural code of the retina, Neuron 22 (1999) 435–450.

[15] M. Livingstone, D. Hubel, Segregation of form, color, movement, and depth: anatomy, physiology, and perception, Science 240 (1988) 740–749.

[16] A. Derrington, P. Lennie, Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque., The Journal of physiology 357 (1984) 219–240.

[17] S. Kristensen, F. E. Garcea, B. Z. Mahon, J. Almeida, Temporal frequency tuning reveals interactions between the dorsal and ventral visual streams, Journal of cognitive neuroscience 28 (2016) 1295–1302.

[18] J. Shlens, G. D. Field, J. L. Gauthier, M. I. Grivich, D. Petrusca, A. Sher, A. M. Litke, E. Chichilnisky, The structure of multi-neuron firing patterns in primate retina, The Journal of neuroscience 26 (2006) 8254–8266.

[19] J. Shlens, G. D. Field, J. L. Gauthier, M. Greschner, A. Sher, A. M. Litke, E. Chichilnisky, The structure of large-scale synchronized firing in primate retina, The Journal of Neuroscience 29 (2009) 5022–5031.

[20] T. R. Vidyasagar, K. Pammer, Dyslexia: a deficit in visuo-spatial attention, not in phonological processing, Trends in Cognitive Sciences 14 (2010) 57 – 63.

[21] Dimigen, O., Sommer, W., Hohlfeld, A., Jacobs, A., & Kliegl, R. (2011). Coregistration of eye movements and EEG in natural reading: Analyses & Review. Journal of Experimenta Psychology: General, 140 (4), 552-572

[22] Dataset [Online]. Available: www2.hu-berlin.de/eyetracking-eeg

[23] Delorme, A., & Makeig, S. (2004). Eeglab: an open source toolbox for analysis of single-trial eeg dynamics including independent component analysis. Journal of neuroscience methods, 134(1), 9–21.

[24] Delorme, A., Sejnowski, T., & Makeig, S. (2007). Enhanced detection of artifacts in eeg data using higher-order statistics and independent component analysis. Neuroimage, 34(4), 1443–1449.

[25] Yuval-Greenberg, S., Tomer, O., Keren, A. S., Nelken, I., & Deouell, L. Y. (2008). Transient induced gamma-band response in EEG as a manifestation of miniature saccades. Neuron, 58(3), 429-441.

[26] Keren, A. S., Yuval-Greenberg, S., & Deouell, L. Y. (2010). Saccadic spike potentials in gamma-band EEG: characterization, detection and suppression. Neuroimage, 49(3), 2248-2263.

[27] D. Manolakis, V. Ingle, Applied Digital Signal Processing: Theory and Practice, Cambridge University Press, pp. 400–403.

[28] S. A. Burns, A. E. Elsner, Response of the retina at low temporal frequencies, JOSA A 13 (1996) 667–672.

[29] E. Castet, Perception of intra-saccadic motion, in: Dynamics of visual motion processing, Springer, 2009, pp. 213–238.

[30] T. V. Sewards, M. A. Sewards, Alpha-band oscillations in visual cortex: part of the neural correlate of visual awareness?, International journal of psychophysiology 32 (1999) 35–45.

[31] I. Fr¨und, N. A. Busch, U. K¨orner, J. Schadow, C. S. Herrmann, EEG oscillations in the gamma and alpha range respond differently to spatial frequency, Vision Research 47 (2007) 2086 – 2098.

[32] W. H. Merigan, J. H. R. Maunsell, How parallel are the primate visual pathways?, Annual Review of Neuroscience 16 (1993) 369–402.

[33] M. C. M. Bastiaansen, L. Magyari, P. Hagoort, Syntactic unification operations are reflected in oscillatory dynamics during on-line sentence comprehension., J. Cognitive Neuroscience 22 (2010) 1333–1347.

[34] I. Smyrnakis, V. Andreadakis, V. Selimis, M. Kalaitzakis, T. Bachourou, G. Kaloutsakis, G. D. Kymionis, S. Smirnakis, I. M. Aslanides, Radar: A novel fast-screening method for reading difficulties with special focus on dyslexia, PloS one 12 (2017) e0182597.

[35] D. Manolakis, V. Ingle, Applied Digital Signal Processing: Theory and Practice, Cambridge University Press, pp. 296–297.

[36] E. Castet, G. S. Masson, Motion perception during saccadic eye movements, nature neuroscience 3 (2000) 177–183.

[37] D. C. Burr, M. C. Morrone, J. Ross, Selective suppression of the magnocellular visual pathway during saccadic eye movements, Nature 371(1994) 511–513.

[38] A. Thiele, P. Henning, M. Kubischik, K.-P. Hoffmann, Neural mechanisms of saccadic suppression, Science 295 (2002) 2460–2462.

AUTHORS PROFILE

Parameswari Shunmugam is a PhD candidate at Universiti Malaya. She has a BA from Universiti Kebangsaan Malaysia, and a MEd from Universiti Putra Malaysia. Her research interests include education, learning disabilities, EEG, fMRI

Vijayakumar is a Senior Lecturer at Multimedia University, Cyberjaya, Malaysia. He holds a BSc from NUS, MSc from UCL and a PhD from MMU, Malaysia. His research interests include signal processing,and image processing. He works mainly with biomedical signals and images.

Nachiappan Murugavadigal is a Professor of Neurosurgery at the Department of Surgery, Melaka Manipal Medical College, 75150 Melaka, Malaysia.

He obtained his MBBS from Karnataka University and and FRCS(Scotland). His research interests include Stereotactic Surgery, Medical Education among others.

Kalai Arasu Muthusamy is a Neurosurgeon at Universiti Malaya Medical Centre and an Assoc Professor at the Medical Faculty, Universiti Malaya. He obtained his MBBS from Universiti Malaya and a PhD from the University of Oxford. His research interests include EEG, DTI, Parkinson’s Disorder among others.