



Perisaccadic Gamma Modulation in Parkinson Disease Patients and Healthy Subjects

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INTRODUCTION

“Low” gamma frequency range (35 - 45 Hz) EEG is recordable over wide region of the scalp in humans. Forgascs et al (2008) et al studied the temporal course of gamma synchrony associated with a voluntary saccade. Gamma power increases over parieto-occipital sites with the beginning of a voluntary eye movement. It reaches its peak (Gamma Modulation Index = $gMI = \text{Peak Gamma}/\text{Mean Gamma}$) before the eyes fixate. Perisaccadic gamma EEG has not been studied previously in PD patients. We have quantified perisaccadic gamma modulation for voluntary saccades in PD patients. Our purpose was to evaluate if perisaccadic gamma correlates with saccade metrics in PD.

PURPOSE

- To evaluate and compare in the PD patients and healthy subjects:
- (1)-Quantify perisaccadic EEG modulation in the gamma and beta range (18-24Hz).
 - (2)-Does gMI lateralize with saccade direction?(centripetal=towards body midline AND centrifugal=away from body midline/center).
 - (3)-is there a correlation between peri saccadic GM and saccade metrics in Parkinson disease?

METHODS

The EEG was recorded with Electro-cap, over the frontal, parietal, temporal and occipital sites in 12 healthy subjects (age 23-30 yrs, 3 females) and in 5 PD patients (age 60-70 yrs, 2 females). Hoehn and Yahr stage ranged 2-3. We used EOG and ISCAN camera for recording the eye movements. Subjects executed saccades to a mark at right on a screen and back to fixation point/midline and vice versa. 2 minutes of EEG were obtained from each subject for each of the eight possible saccades (rightwards and leftwards, centripetal and centrifugal x 2 distances: 15 and 30 degrees). Perisaccadic EEG segments of 1 sec time window were selected for offline analysis. We used continuous wavelet transformation and Hilbert transform to quantify the perisaccadic Gamma and Beta powers, for individual saccades. The power results for all saccades were averaged to obtain an average gamma and beta power for all eight types of saccades. The wavelet we selected was a complex Gaussian filter. General linear mixed model was used for statistical analysis.

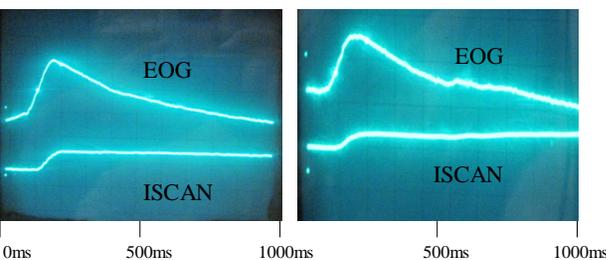


Figure 1, 2. illustration of an average of 25 voluntary rightwards saccades towards their representative center/body midline, at a 30 degree distance, in a healthy subject (left) and a PD patient (right). Increase in voltage is in micro V (y axis) as a function of time in ms (x axis). PD patients make step saccades in reaching towards the visual target.

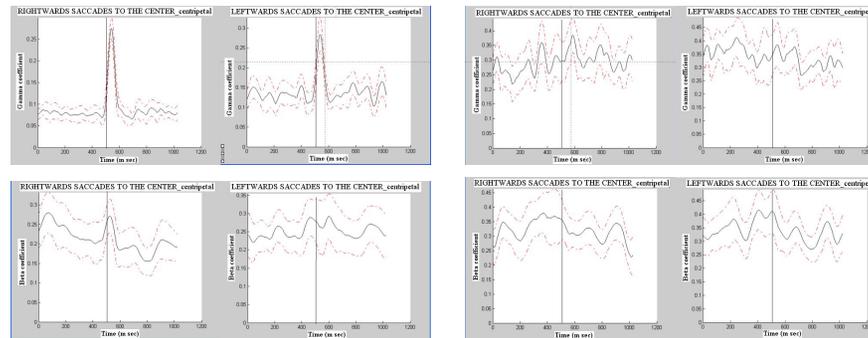


Figure 3 - 10. Average gamma (top) and beta power (bottom) as a function of time (ms) as healthy subject (left side) and PD patient (right side). The traces were obtained over left and right lateral post. parietal electrode sites. Vertical line at 500 ms represents start of saccade. Significant gamma increase (gMI) is seen intrasaccadically in healthy subjects compared to PD patient. There is no prominent intra saccadic modulation of Beta range EEG in PD or in healthy subjects.

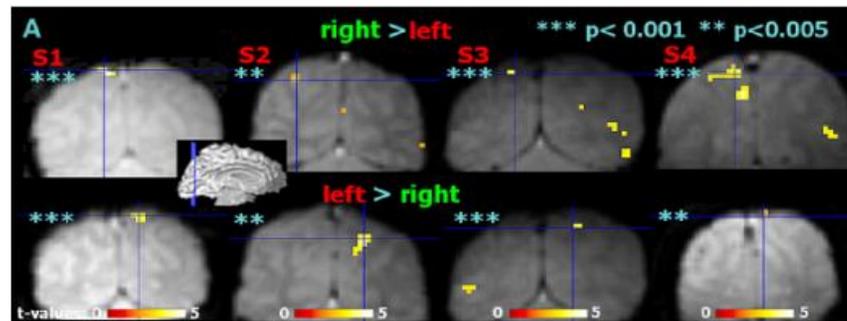


Figure 11. BOLD imaging of parietal cortical activity contralateral to saccade direction in healthy subjects (Rieger et al, 2008_reference 2). Upper row shows where leftwards saccades elicit higher BOLD response than rightwards saccades and lower row depicts the results of reverse comparison. Stars indicate uncorrected p value thresholds applied to images. Color bar indicates t value code.

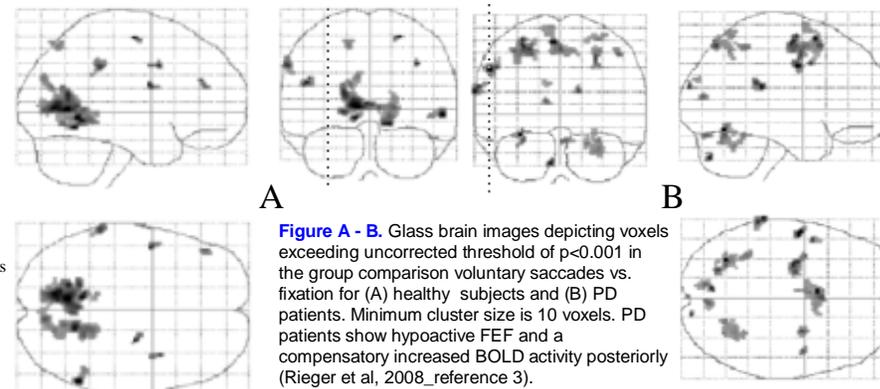


Figure A - B. Glass brain images depicting voxels exceeding uncorrected threshold of $p < 0.001$ in the group comparison voluntary saccades vs. fixation for (A) healthy subjects and (B) PD patients. Minimum cluster size is 10 voxels. PD patients show hypoactive FEF and a compensatory increased BOLD activity posteriorly (Rieger et al, 2008_reference 3).

RESULTS

1. Modulation of perisaccadic gamma as a function of saccade direction is prominent in healthy subjects. gMI is higher over the hemispheres contralateral to saccade direction, both towards and away from midline, for anterior parietal ($p < 0.005$), posterior parietal ($p < 0.011$) and occipital ($p < 0.011$) sites. Unlike healthy subjects, PD patients do not show a prominent intrasaccadic gMI.
2. No prominent perisaccadic beta modulation is evident in PD patients or in healthy subjects.
3. Towards center vs. away from center saccades: gMI is significantly higher for saccades to the midline compared to those away from body center in healthy subjects.

DISCUSSION / CONCLUSION

Our results show an association of impaired saccade kinetics in PD with lack of intrasaccadic gamma power modulation. PD patients have hypometric voluntary saccades in which the primary saccade undershoots followed by small corrective saccades (multi steps). Previous functional MRI studies (Rieger et al, 2008) show that the PD patients, unlike the healthy subjects, have inactive FEF as they make horizontal voluntary saccades towards a target. FEF is the motor area for saccade initiation. The anatomical evidence shows perisaccadic frontal hypofunction in PD (Fig A, B) while the EEG shows impaired gamma modulation. We suggest that FEF is involved in sending signals to parietal cortex (PEF), directly or in the form of a corollary discharge, thereby causing intrasaccadic neuronal reorganization (gamma range oscillation) in the parieto-occipital cortex.

REFERENCES

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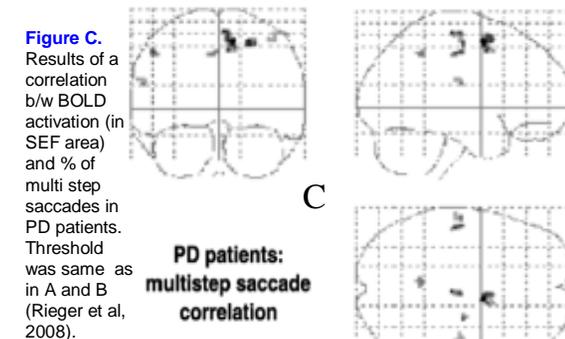


Figure C. Results of a correlation b/w BOLD activation (in SEF area) and % of multi step saccades in PD patients. Threshold was same as in A and B (Rieger et al, 2008).