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Objective

Alzheimer's disease (AD) is the major cause of dementia in the middle-age and elderly, manifesting with progressive impairment of memory, spatial, linguistic and attentive functions. Visual system abnormalities are frequent. Increased saccadic latency, saccadic intrusions (square-wave jerks type, SWJs) and a higher rate of errors in the antisaccade task are the most common oculomotor changes described, while dynamic anomalies are infrequent. Here we report on the progression of oculomotor abnormalities in a probable AD patient.

Materials and Methods

A 81-years-old man, presenting with progressive memory loss associated with difficulties in writing and reading since 3 years, underwent clinical, laboratory, neuropsychological and neuroimaging (MRI and PET-FDG scan) examination. The oculomotor tasks were recorded with eye-tracking technique during patient's first examination and nine months later. Standard saccadic parameters of visually-guided saccade and antisaccades were carried out. The rate of antisaccade errors with relative corrections and fixation abnormalities were also evaluated.

Results

At the time of first observation his MMSE was 19,4/30. MRI showed parietal-temporal-insular cortical atrophy; PET-FDG scan evidenced an hypometabolism in the posterior left parietal region and lateral and mesial left temporal cortex. Based on the results of the investigation, a diagnosis of probable Alzheimer's disease was considered (NINCDS-ADRDA criteria). Visually-guided saccades examination showed increase of saccade latency and slight hypometria. The antisaccade error rate was 71,8% with correction rate of 60%. Saccadic intrusions (SWJs subtype) were detected during steady fixation. Nine months later, MMSE reduced to 16.4/30; patient started complaining about visual agnosia. Saccadic performance worsened, with a further increase in latency, he was unable to perform the antisaccade task and the SWJs frequency increased to 70/min.

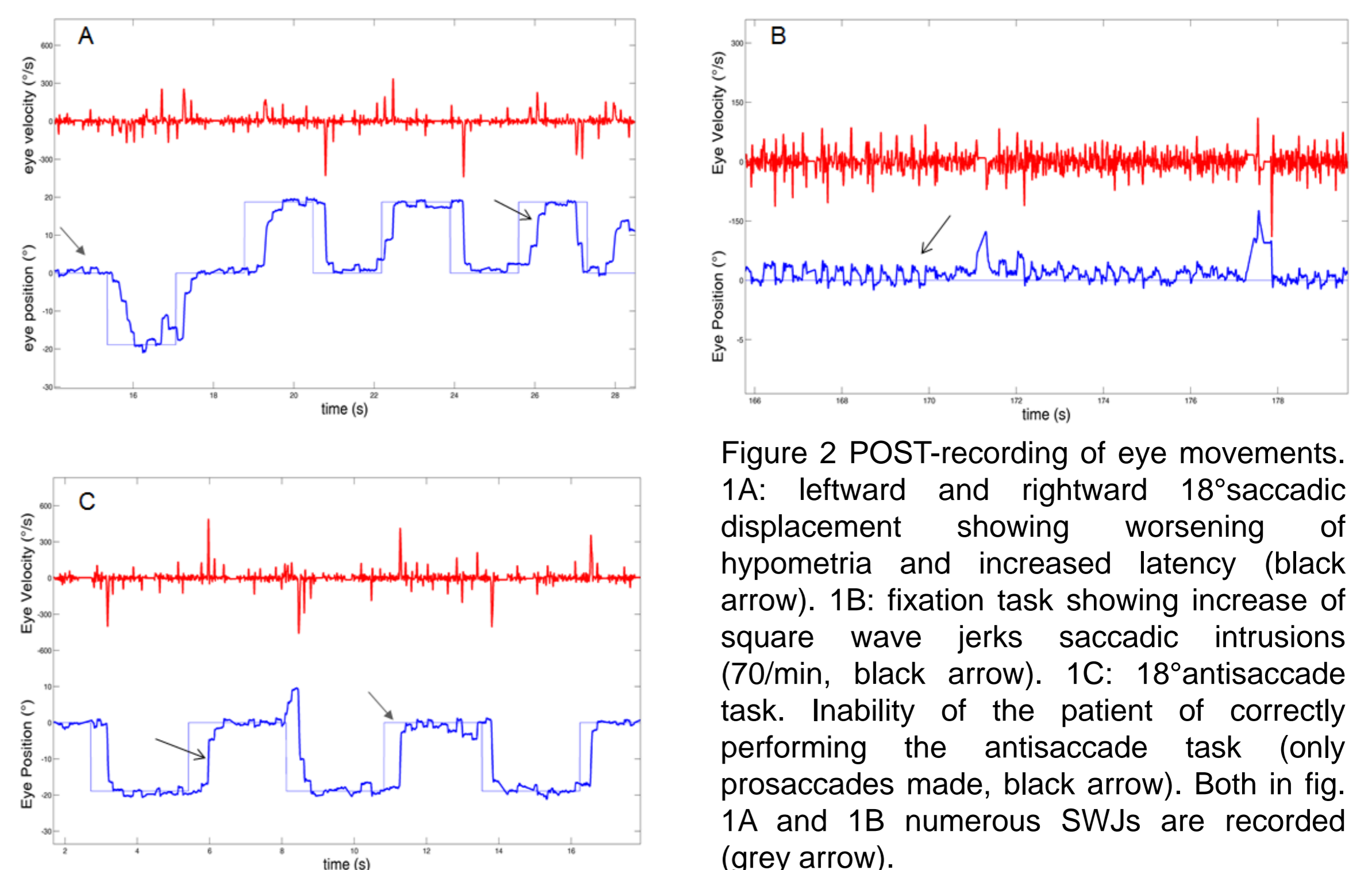


Figure 2 POST-recording of eye movements. 1A: leftward and rightward 18°saccadic displacement showing worsening of hypometria and increased latency (black arrow). 1B: fixation task showing increase of square wave jerks saccadic intrusions (70/min, black arrow). 1C: 18°antisaccade task. Inability of the patient of correctly performing the antisaccade task (only prosaccades made, black arrow). Both in fig. 1A and 1B numerous SWJs are recorded (grey arrow).

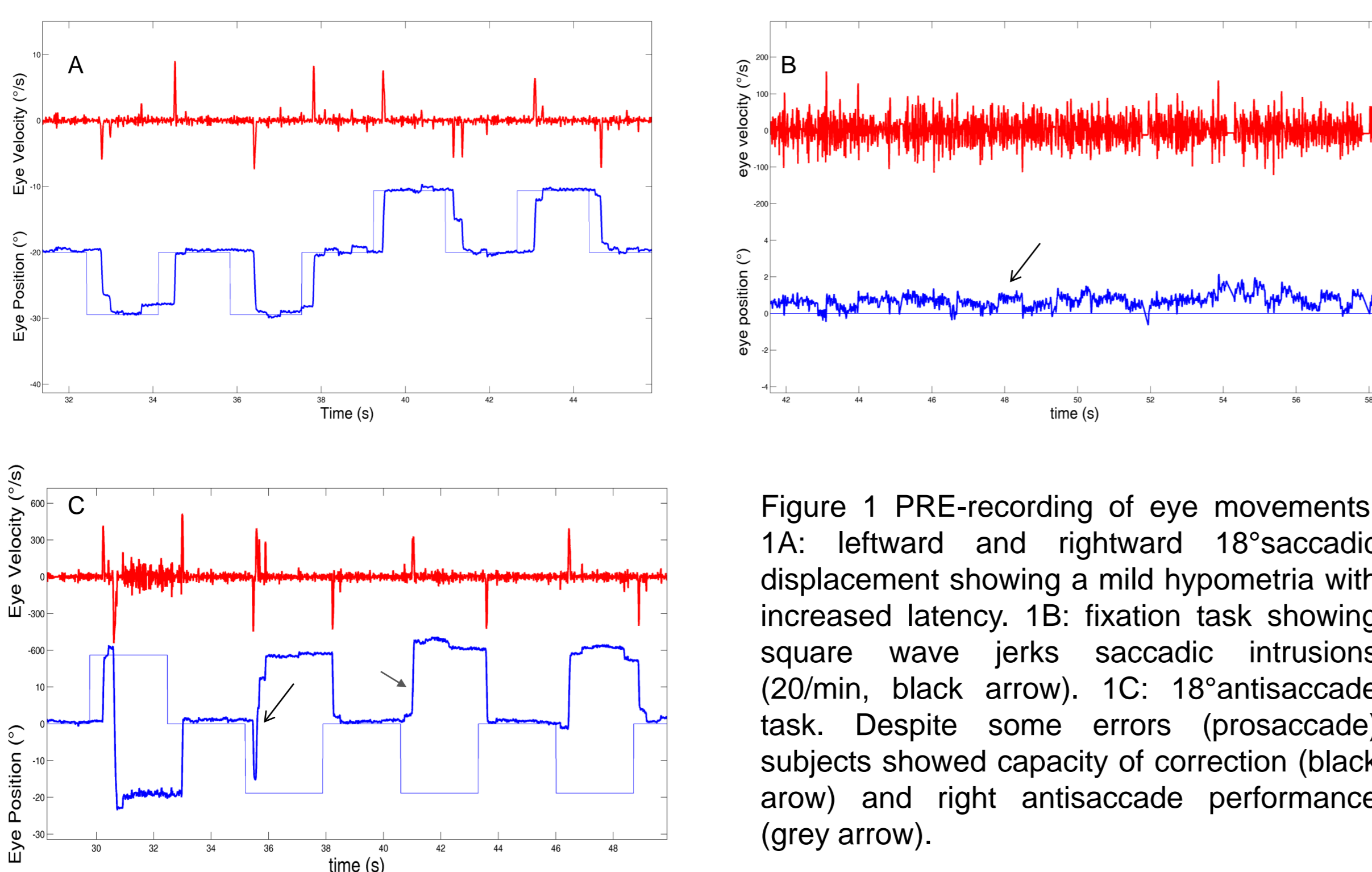


Figure 1 PRE-recording of eye movements. 1A: leftward and rightward 18°saccadic displacement showing a mild hypometria with increased latency. 1B: fixation task showing square wave jerks saccadic intrusions (20/min, black arrow). 1C: 18°antisaccade task. Despite some errors (prosaccade) subjects showed capacity of correction (black arrow) and right antisaccade performance (grey arrow).

Table 1. Saccadic parameters of patient vs controls.

Saccadic values	Latency	Peak Velocity	Amplitude
	10° (265±98)ms	10°: (312±60)°/s	10° (8,7±1,4)°
Pre	18° (282±91)ms	18°: (436±51)°/s	18° (16,9±1,7)°
	8° (289±46)ms	8°: (232±71)°/s	8° (7,4±2,2)°
	10° (285±80)ms	10°: (285±57)°/s	10° (8,8±2,4)°
Post	18° (288±91)ms	18°: (336±99)°/s	18° (15,3±3,8)°
	8° (357±73)ms	8°: (129±36)°/s	8° (5,2±0,9)°
	10° (178±21)ms	10° (388±60)°/s	10°: (10,3±1)°
Ctrl	18° (189±44)ms	18° (507±78)°/s	18°: (18±1)°
	8° (199±17)ms	8° (289±42)°/s	8° (8±0,8)°

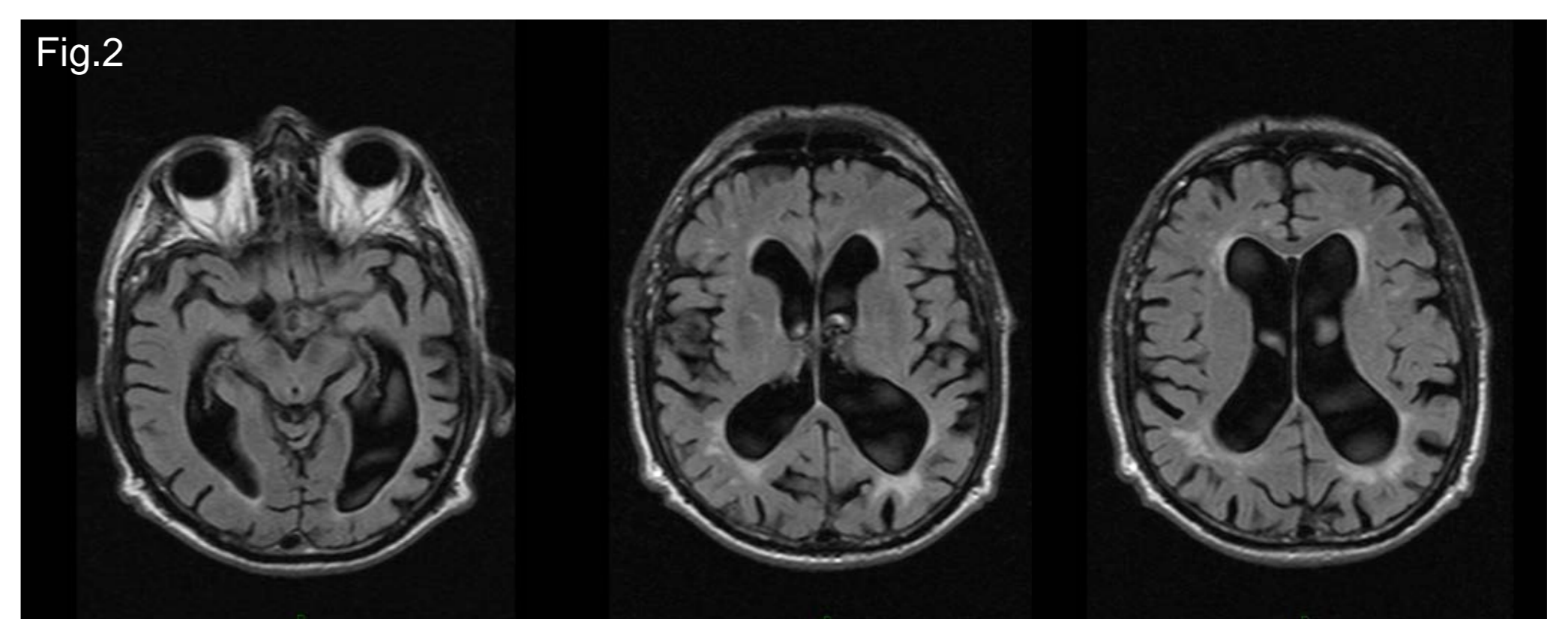


Fig. 2 Brain MRI (PRE) showing a marked cortical atrophy, particularly of parietal-temporal-insular regions, and aspecific periventricular white matter abnormalities.

Discussion

Eye movement abnormalities are a well-known finding in AD but their prevalence and correlation with disease progression are still controversial. Increase of saccadic latency with slight hypometria was the first abnormality during the first examination, while the frequency of saccadic intrusions resembled that of controls; despite the high number of antisaccade errors, the patient showed a good capacity of correction. After 9 months, all the examined parameters worsened; particularly, the patient lost completely the ability to correct antisaccade errors and his fixation was interrupted by a high frequency of SWJ. Worsening of these specific oculomotor parameters paralleled the MMSE reduction, confirming the progression of cognitive impairment. Our findings testify the presence of specific oculomotor abnormalities in a probable AD patient, correlated with disease progression. Saccadic evaluation represents a non-invasive, repeatable tool in the early screening and follow-up of dementia.