The role of the cerebellum in the antisaccade performance: insights from Spinocerebellar Ataxia Type 2 and Late Onset Cerebellar Ataxia

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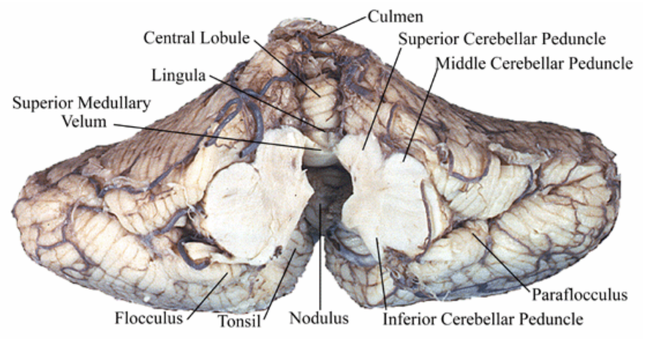
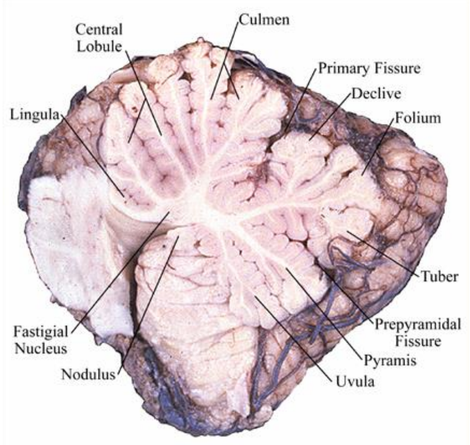
INTRODUCTION

The medial cerebellum is the brain structure where the motor command for saccades is optimized by means of gain control, accuracy preservation and endpoint error minimization. However recent studies have suggested an involvement of the lateral cerebellum in complex saccadic behaviour.

In order to further explore the role of the cerebellum in the cognitive aspects of motor behaviour we studied the antisaccadic performance in two types of cerebellar pathology: Late Onset Cerebellar Ataxia (LOCA), a model of isolated cerebellar dysfunction, and Spinocerebellar Ataxia type 2 (SCA2) in which neurodegeneration involves also other structures including the brainstem burst generator of saccades.

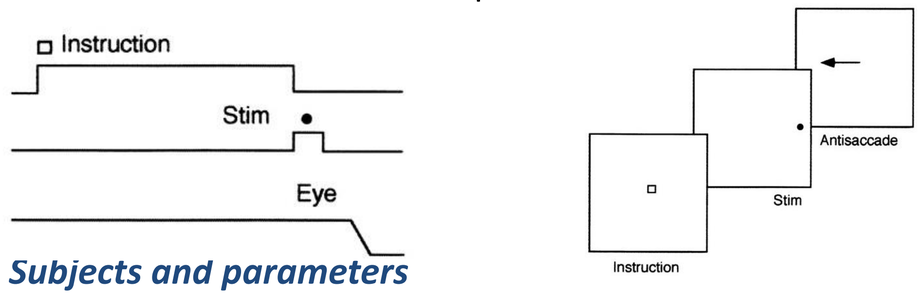




METHODS

Tasks and eye movements recording

Data were obtained using an infrared eye-tracker system (ASL 6000). A 9-point calibration was performed before each recording. The visual stimulus was presented on a 31x51 cm LCD screen. Sampling frequency was 240Hz. The Antisaccade task at ±10 and ± 18° was performed.

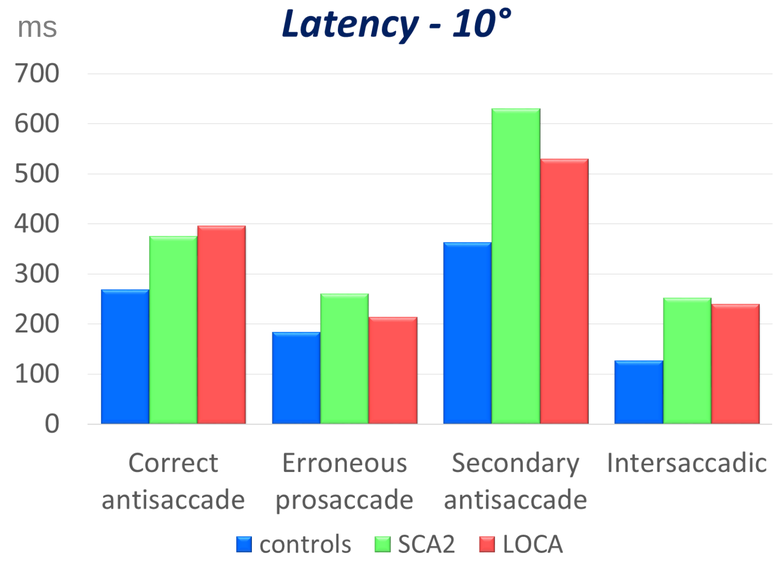


Subjects and parameters

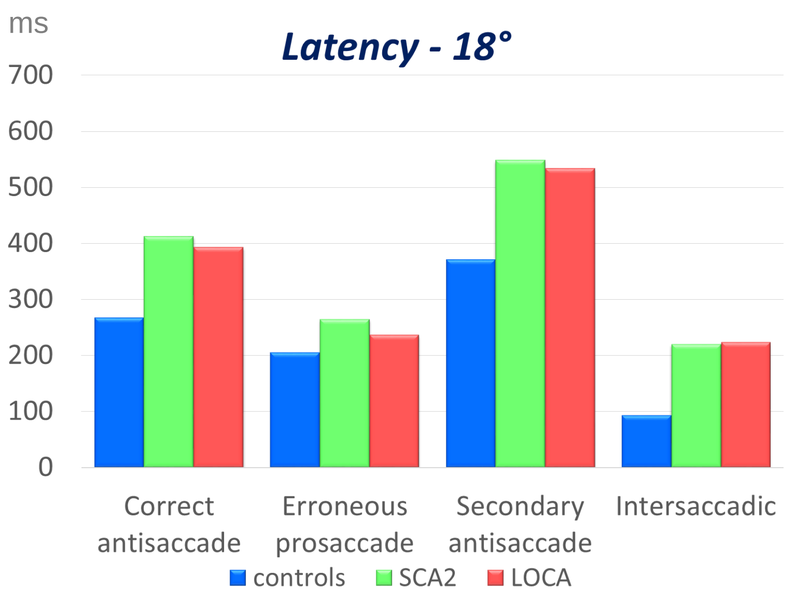
Latency, duration, peak and mean velocity, amplitude, gain and absolute error of antisaccades were calculated in addition to erroneous prosaccades, corrective antisaccades and "intersaccadic" latencies, separately at 10 and 18 in 12 SCA2 patients, 10 LOCA patients and 45 healthy controls

RESULTS

Latency - 10°



Latency - 18°



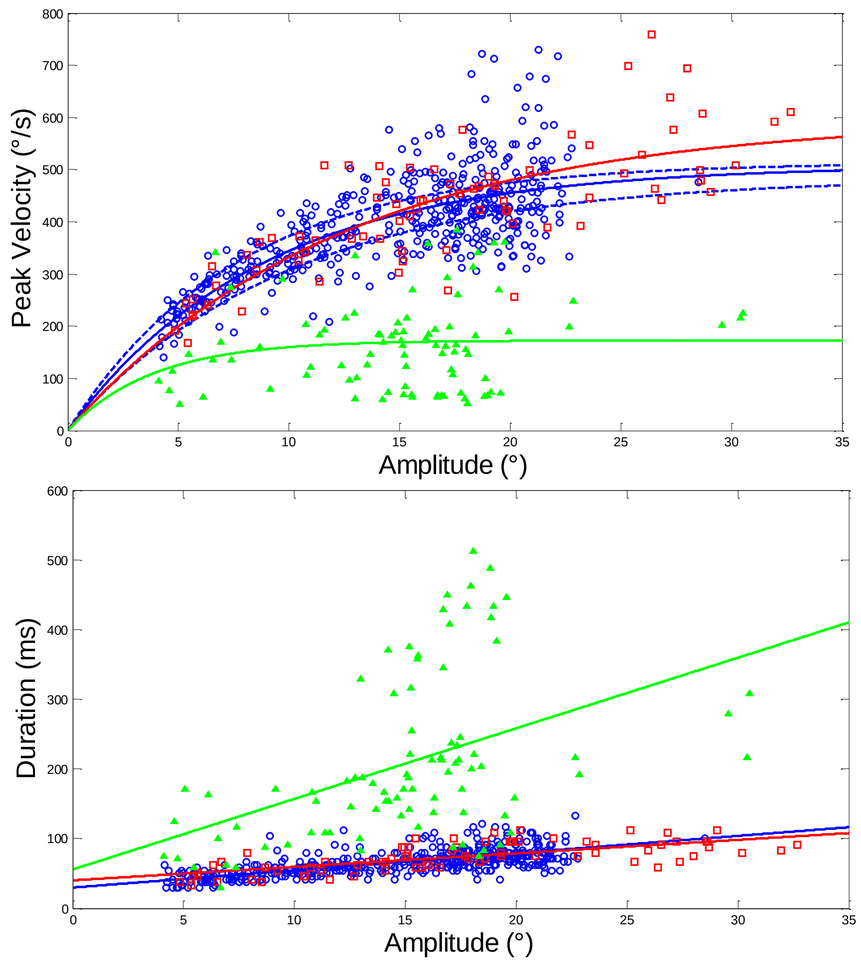
Correct antisaccade latency
10°: SCA2 > controls (P<0.05) and LOCA > control (P<0.001)
18°: SCA2 > controls (P<0.001) and LOCA > control (P<0.001)

Erroneous prosaccade latency
10°: no significant differences
18°: SCA2 > controls (P<0.05)

Secondary antisaccade latency
10°: SCA2 > controls (P<0.001) and LOCA > control (P<0.05)
18°: SCA2 > controls (P<0.05) and LOCA > control (P<0.05)

Intersaccadic latency
10°: SCA2 > controls (P<0.05) and LOCA > control (P<0.05)
18°: SCA2 > controls (P<0.05) and LOCA > control (P=0.001)

Correct antisaccade dynamic parameters



Duration
10°: SCA2 > controls (P<0.001) and LOCA (P<0.05)
18°: SCA2 > controls (P<0.001) and LOCA (P<0.001)

Peak velocity
10°: SCA2 < LOCA (P<0.05); LOCA > controls (P<0.05)
18°: SCA2 < controls (P<0.001) and LOCA (P<0.001)

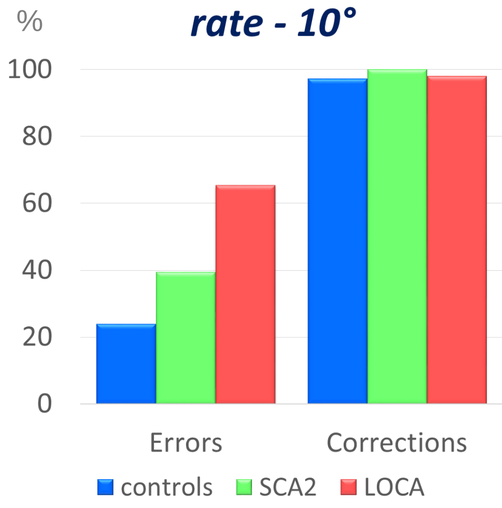
Mean velocity
10°: SCA2 < LOCA (P<0.001) and controls (P<0.05)
18°: SCA2 < LOCA (P<0.001) and controls (P<0.001)

Amplitude
10°: SCA2 > controls (P<0.05) and LOCA > controls (P<0.001)
18°: no significant differences

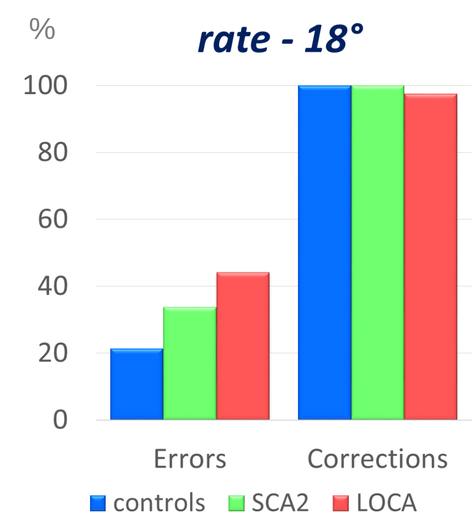
Gain
10°: SCA2> controls (P=0.001) and LOCA > controls (P<0.001)
18°: SCA2> controls (P<0.05)

Absolute error
10°: no significant differences
18°: SCA2 > controls (P<0.05)

Error and correction rate - 10°



Error and correction rate - 18°



Error rate
10°: SCA2>cntr (p=s), LOCA>cntr (p=s), LOCA>SCA2 (p=s)
18°: SCA2>cntr (p=s), LOCA>cntr (p=s)

Correction rate
10°: no significant differences
18°: no significant differences

CONCLUSIONS

Our study shows an increased latency of correct and secondary antisaccades in both groups of patients, suggesting a role of the cerebellum in the timing of the planning of an antisaccadic movement. Whether this delay reflects a prolonged accumulation rate towards the execution of an antisaccade instead of a prosaccade or a defect in visual vector inversion process, or both, is unknown. Moreover, the high number of errors in the antisaccadic task observed in both groups of patients strongly supports an involvement of cerebellum in motor programming and cognitive voluntary behaviour.

In conclusion, our results are in line with the hypothesis of a role of the cerebellar structures in voluntary behaviour by the inhibition of unnecessary reflexive movements and the facilitation of goal directed actions.

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