



The PSP Association's International Medical Workshop 7th July 2009

ABSTRACT

Title of Talk: SODIUM VALPROATE FOR SLOWING DOWN DISEASE PROGRESSION IN PROGRESSIVE SUPRANUCLEAR PALSY. A FRENCH MULTICENTER RANDOMIZED PLACEBO-CONTROLLED STUDY

Part 1: Speaker(s) details

Title	Professor
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Position	

Part 2: Abstract (Maximum 400 words) Please make your abstract easy to understand as it will appear on our website and will be read by people with PSP and their carers who are not scientists but who will want to understand your work and what it means for them.

Background. Hyperphosphorylation of tau protein might be involved in the abnormal aggregation observed in the brain of patients suffering from progressive supranuclear palsy (PSP). The anticonvulsant drug sodium valproate (VPA) is an inhibitor of glycogen synthase kinase 3 β , one of the main kinases responsible for tau phosphorylation. Reduction of tau phosphorylation might thus lead to a slowing down of neurodegeneration in PSP.

Objective. To assess the efficacy of VPA for slowing down PSP progression.

Study design. Multicenter randomized double-blind placebo-controlled trial.

Method. Patients had been randomly assigned to treatment of a daily dose of 1,500 mg VPA (500 mg/week titration) or to placebo. The primary outcome measure is the change in score of the PSP Rating Scale after 24 months of treatment. Other outcome measures are the Mattis Dementia Rating Scale, the Minimental Test, various scales for frontal lobe assessment and the Neuropsychiatric Inventory.

Preliminary results. Inclusion started in November 2006. The recruitment period is finished. Twenty-eight patients had been included. Twenty-one had finished the 24-month period of treatment, one stopped due to a delirium, six are still in the treating period. The last visit for the last patient will be in July 2010. Eight patients developed sleepiness and delirium especially during the first weeks of treatment, but all but one were able to receive 1,500 mg of VPA or placebo daily. Hallucination was observed in two patients, a side-effect that was resolved after adjustment of the antiparkinsonian drug treatment (withdrawal of amantadine).

Conclusion. Several lines of evidence support the putative interest of VPA as a means of slowing down disease progression in PSP. A multicenter placebo-controlled trial is being conducted to test this hypothesis. The results of the study are expected by the end of 2010.