P3-322 PREVIOUS ERRORLESS SEQUENCE-LEARNING PROMOTES SUBSEQUENT SRT PERFORMANCE IN PATIENTS WITH ALZHEIMER'S DISEASE

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Background: Motor-learning capacities are known to be relatively preserved in Alzheimer's disease (AD), which is crucial in the context of the patient's autonomy (e.g., Rouleau et al., 2002). However, it is important to determine the most appropriate techniques for such learning. In AD, implicit or procedural rehabilitation techniques would be more effective to train new skills than explicit or declarative learning methods (van Halteren-van Tilborg, 2007). Maxwell et al. (2001) showed that reducing errors during motor learning minimizes the building of declarative knowledge and would allow implicit knowledge accumulation. If errorless learning induces the formation of an implicit knowledge, this technique appears to be adapted to the learning of a perceptual-motor skill in patients with impaired controlled processes. Very few studies have investigated errorless learning in procedural learning situations, even though some data suggest that errorless learning would be efficient for learning instrumental activities of daily living (e.g., Thivierge et al., 2008). **Methods:** In this study we examined the acquisition of a new perceptual-motor skill in 12 patients with AD and 12 healthy older adults. We compared the impact of two preliminary sequence learning conditions (errorless vs. errorful) on a serial reaction time (SRT) performance. In SRT, the subject must react as quickly as possible to the appearance of a target on a screen by pressing the key corresponding to the position of the stimulus. The effectiveness of learning is demonstrated by a reaction time improvement when the target follows a repeating sequence. **Results:** For patients with AD, results confirm that the advantage provided by prior learning occurs only in the errorless condition (P=0,05) whereas both learning modes improve SRT performance in healthy participants ($P = 0.02$ for errorless learning and $P<0.01$ for errorful learning). Moreover, by using the process dissociation procedure (Jacoby, 1991), we show that performance on a subsequent generation task was more sustained by controlled processes for the errorful condition than for the errorless learning (P=0.03). **Conclusions:** In conclusion, these results confirm that the errorless learning promotes the development of implicit knowledge and appears to be an effective method for procedural learning in Alzheimer's disease.

P3-323 INTERDISCIPLINARY INTERVENTION DECREASES COGNITIVE IMPAIRMENT FOR OLDER TAIWANESE INDIVIDUALS WITH HIP FRACTURE: TWO-YEAR FOLLOW-UP

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Background: Cognitive impairment, which occurs in 31-88% of postoperative elderly persons with hip fracture, has been found to predict poor functional recovery and to increase risk of mortality. However, few studies on more long-term cognitive functioning of these patients have been conducted. The purpose of the study was to explore the 2-year postoperative trajectory for cognitive function of hip-fractured elders and cognitive effects of an interdisciplinary intervention. **Methods:** Of 160 subjects randomly assigned to groups, 29 (35.8%) in the control group (n = 81) and 30 (38.0%) in the intervention group (n = 79) were cognitively impaired at admission. Intervention group was received geriatric consultation, continuous rehabilitation, and discharge planning. Subjects' cognitive function was measured using the mini-mental state examination, Taiwan version at admission, 6, 12, 18 and 24 months after discharge. **Conclusions:** Our interdisciplinary intervention improved the long-term postoperative cognitive functioning of elderly persons with hip fracture in Taiwan. Postoperative cognitive impairment will need to be clinically assessed and managed for at least 2 years following discharge.

P3-324 BETA-AMYLOID NEUROTOXICITY AND NEUROPROTECTIVE ROLE OF 17β ESTRADIOL IN AGING RAT BRAIN SYNAPTOSOMES

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Background: Alzheimer's disease (AD) is the most common form of dementia in the elderly. AD is characterized by the presence of amyloid plaques which are formed from deposits of beta-amyloid protein (Aβ). Accumulation of oligomeric Aβ in the brain contributes to neuronal dysfunction and ultimately leads to neurodegeneration. During aging the brain experiences structural, molecular, and functional alterations. These changes increase during menopausal condition in females when the level of estradiol is decreased. The aim of the present study was to determine the effect of neuropeptide, neurokinin B (NKB) and beta-amyloid fragment Aβ (25-35) on 17β estradiol (E2) treated aging female rat brain of 3 months (young), 12 months (adult) and 24 months (old) age groups. **Methods:** The aged rats (12 and 24 months old) (n= 8 for each group) were given subcutaneous injection of 17 b -estradiol (0.1 µg/g body weight) daily for one month. After 30 days of hormone treatment, experimental animals of all the groups were sacrificed and brains were isolated for further study. Aging brain function were assayed by measuring the activities of antioxidant enzymes, monoamine oxidase (MAO), membrane bound ATPases, intracellular calcium levels and lipid peroxidation in presence of neuropeptides. **Results:** The results obtained in the present work revealed that i ncreased activities of antioxidant enzymes, membrane bound ATPases and decrease in level of calcium levels, MAO actiivty and lipid peroxidation in presence of NKB and combined NKB and Aβ *in vivo* estradiol (E2) treated ageing rat brain. NKB treatment reversed the beneficial in preventing some of the age related changes in the brain. An *in vitro* incubation of E2 treated synaptosomes with Aβ showed toxic effects on all the parameters, while NKB showed stimulating effects and the combined NKB and Aβ showed a partial effects as compared to Aβ (25-35) and NKB alone. **Conclusions:** Present study elucidates an antioxidant, neuromodulatory and neuroprotective role of tachykinin peptide NKB against the beta amyloid induced toxicity in E2 treated female rats. NKB treatment reversed the beneficial in preventing some of the age related changes in the brain.

P3-325 VACCINATION AGAINST TAU IMPEDES PROGRESSION OF NEUROFIBRILLARY HISTOPATHOLOGY IN AGED P301L TAU TRANSGENIC MICE

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Background: In Alzheimer's disease (AD) brains, the microtubule-associated protein tau and beta-amyloid (Aβ) deposit as intracellular neurofibrillary tangles (NFTs). Tau deposits are furthermore found in a significant number of frontotemporal dementia (FTD) cases. These diseases are characterized by progressive neurodegeneration, the loss of intellectual capabilities and behavioral changes. Unfortunately, current therapeutic approaches are limited. Active immunization against pathogenic tau has only recently been shown to prevent pathology in young tau transgenic mice, when treated before the onset of disease. However, in humans, diagnosis and treatment would be routinely done when symptoms are overt, meaning that the histopathological changes are already present. **Methods:** We used active immunization