Sensorimotor control and proprioception in neurorehabilitation

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Introduction

It is widely assumed that changes in sensorimotor control are strongly connected with changes in proprioceptive performance. This assumption is based on a couple of injury and disease studies. Thus disturbances in gait and posture as well as impairments in proprioception are found in Parkinson’s disease (Rickards / Cody 1997, Khudados et al. 1999, Maurer et al. 2003). Other experiments found that artificial proprioceptive stimulation leads to motor control errors (Verschueren et al. 1999a, b). It was widely suggested that this connection is also valid the other way round i.e. improved sensorimotor control is based on higher proprioceptive performances. However regarding to alternative neurophysiological theories we question this strong connection.

Methods

25 physical education students and 25 Parkinson’s disease patients participated in this study. Both groups were subdivided in an experimental and a control group. Treatment was based on 5 series of random whole body vibration taking 60 seconds each. In earlier studies it was shown that this treatment leads rapidly to postural control improvements (Haas et al. 2004a, b). Pre and post treatment proprioceptive capabilities were tested using a specially constructed device. The angle of the knee joint was measured using a goniometer. Via knee extension and flexion movements subjects should reproduce a given, slowly oscillating course which was presented at a computer screen. No feedback concerning the success of the task was available. Before tests were started all subjects performed some training sessions to ensure that they were able to reproduce the course precisely.

Results

Figure 1 shows related postural control improvement in PD and an example proprioception analysis, figure 2 presents averaged movement amplitude values of both treatment groups in pre and post tests.

On average undershooting errors at both endpoints can be found in the experimental groups. This leads to lower but insignificant movement amplitudes compared to the given amplitude. The control groups present similar results which do not differ from the experimental group. Pre-post comparison shows also no significant differences in any parameter i.e. amplitude, endpoints, frequency.

Discussion/Conclusion

As the treatment is known to improve postural control strongly in PD as well as in healthy subjects and no changes in proprioceptive capabilities can be identified one has to conclude that positive modifications of sensorimotor control can not be explained primarily by an improved perceptive performance. However based on methodical reasons no statements can be given about possible changes in ankle proprioception.

Further more proprioception was tested at relative slow movement velocity; in contrast ballistic neuromuscular activations are necessary to keep up balance. Further studies have to analyse proprioceptive interaction structures and to identify other mechanism that might explain sensorimotor control improvements respectively. We speculate that supraspinal biochemical changes and activation of the supplementary motor area (SMA) have a strong influence on motor control performance and motor learning.

References

Haas CT et al. (2004) Isok Ex Sci: 12-13
Haas CT et al. (2004) Movement Disorders, 19 Suppl. 9, 185
Khudados E et al. (1999) J Neurol Neurosurg Psychiatry;67: 504-510
Rickards C/ Cody FWJ (1997) Brain: 977-990
Verschueren SMP et al. (1999 a, b) Exp Brain Res: 171-181 / 182-192