Neurorehabilitation 1

210
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Neurorehabilitation 1
The post-operative analgesia of the virtual reality using a mirror therapy after total knee arthroplasty
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Background: Mirror therapy has been tried against phantom pain and CRPS.

Objective: To evaluate analgesic effects of mirror therapy using virtual reality after total knee arthroplasty (TKA).

Methods: Patients undertaking uni-lateral TKA was to be included. Patient who could not freely move the contralateral leg, were not enough clear to indicate VAS, or could not look at virtual reality were excluded. Intervention was provided over two weeks to full term group (FT) or one week to half term (HT). Data were collected at post-operative 2nd, 3rd, and 7th week.

Results: 22 patients recruited. VAS while at resting was 40, VAS while moving 52, active ROM of flexion 100°, active ROM of extension -3°, WOMAC index 31, 6 minute walk test 281 M, timed-stands test 24 seconds, the mean time of requested Tridol intravenous injection of 3 at 1st follow-up. VAS while at resting was 9, VAS while moving 30, active ROM of flexion 105°, active ROM of extension 0°, WOMAC index 18, 6 minute walk test 327 M, timed-stands test 21 seconds, the mean time of requested Tridol intravenous injection of 1 at 2nd one. VAS while at resting was 8, VAS while moving 21, active ROM of flexion 135°, active ROM of extension 0°, WOMAC index 11, 6 minute walk test 351 M, timed-stands test 19 seconds at 3rd one.

Conclusions: Mirror therapy using virtual reality may be beneficial for analgesia against TKA.

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211
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Neurorehabilitation 1
Quantitative diffusion tractography imaging assessment in spasticity
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Purpose: To assess structural brain changes with diffusion tractography (DTI) in subjects with spasticity secondary to acquired brain injury, pre and post peripheral botox treatment.

Method: After Bioethics approval,10 consenting adults were studied, 7 with spasticity and 3 controls, with DTI in 32 directions. Spasticity subjects were studied before and 3 months after injection of botox. Controls were studied once. Entered variables were disability assessment scale, fractional anisotrophy (FA) and apparent diffusion coefficient (ADC) maps of corticospinal tracts and corpus callosum. Average values of control and spasticity subjects were compared. Forward stepwise multiple regression analyses were run considering post botox FA and ADC as dependent and pre-treatment values as independent variables.

Results: No ADC changes were seen. Control showed higher FA than spasticity subjects (table 1). FA showed regression for affected CST after botox vs contralateral CST after and affected CST before treatment with R = .99, R² = .99, adjR² = .99, F(4,2) = 330.54 p < .003 (Tables 2, 3, 4).

Discussion: FA is a quantitative DTI variable indicative of tract indemnity which, in this pilot study, showed: 1. Differences between control and spasticity subjects, 2. Regression between post botox FA in the affected side vs contralateral post and ipsilateral pre-treatment values, and 3. FA drop after treatment. Although the former and second findings were expectable, the latter was not, given noted post treatment clinical improvement. Diminished FA might reflect a decreased upper neuron inhibitor tone when spasticity improves. FA drop after peripheral injection of botox is of potential complex interpretation and needs further testing in larger cohorts.
In post-stroke spasticity, functional imaging may demonstrate central effects of botulinum toxin type A (BoNT) therapy. Seven ischemic stroke patients (4 females, 3 males, mean age 58.9) with hand plegia and spasticity were studied following IRB approval. Spasticity was scored according to the modified Ashworth scale (MAS). FMRI examination was performed 3 times: before (W0) and 4 (W4) and 11 weeks (W11) after BoNT. Whole brain fMRI data were acquired during paced passive wrist extension-flexion movements of the plegic hand alternating with rest blocks. Statistical analysis yielded group session-wise statistic maps and paired between-session contrasts, thresholded at corrected significance level of \( p < 0.05 \).

BoNT transiently lowered MAS scores at W4 (mean MAS at W0: 2.2 (SD 0.43), at W4: 1.25 (SD 0.46), at W11: 1.93 (SD 0.12)). In all the sessions, group fMRI activation of the ipsilesional sensorimotor cortex dominated (M1, S1 and SMA). At W4, bilateral cerebellum activation transiently emerged. An additional activation cluster in the contralesional sensorimotor cortex manifested at W0 and W4, but disappeared at W11.

Significant between-session differences: W4 vs. W0 (bilateral cerebellum, occipital cortex, prefrontal cortex) and W4 vs. W11 (ipsilesional cerebellum, contralesional thalamus, bilateral premotor cortex and areas from the default mode network). Activation decreases over time appeared in bilateral parieto-occipital cortex (W0 vs. W4) and bilateral M1, S1, SMA and precuneus (W0 vs. W11).

Whole-brain activation patterns during BoNT treatment of post-stroke arm spasticity and further follow-up demonstrated adaptive changes both within and outside the classical sensorimotor system. Specific BoNT-related changes appeared in the cerebellum.

**Study Support:** IGA MH CR NT13575.
analogue to the Wisconsin Card Sorting Test, which measures executive function in patients with frontal lobe damage, TBI, and psychiatric disorders.

Objective: The goal was to investigate whether exposure to clinically relevant therapies post injury, such as environmental enrichment (EE), an endorsed animal model of neurorehabilitation, and the antidepressant drug, citalopram, a treatment known to alleviate depressive-like symptoms and improve cognition in humans, will attenuate executive function deficits on AST.

Methods: Isoflurane-anesthetized male rats were subjected to a CCI (2.8 mm depth at 4 m/s) or sham injury, and randomly assigned to EE housing and daily injections of citalopram, alone or in combination. At four weeks post-surgery, rats were tested on the AST, involving a series of increasingly difficult discriminative tasks to obtain food reward, including simple and compound discriminations, stimulus reversals, and intra- and extradimensional shifts.

Results: EE and daily citalopram promoted cognitive recovery on the AST after injury when given alone, but more robustly when provided in combination vs. non-EE (p < 0.05).

Conclusions: This combined treatment aims to reflect simultaneous rehabilitation and pharmacotherapies in a clinical setting. Future studies will assess the ideal cognitive recovery timeline and specific brain mechanisms involved in restoring higher function after TBI.

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214  
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Neurorehabilitation 1  
The correlation between the neurological complications of rheumatoid arthritis with the disease activity and functional impairment (Disability)  


Abstract  
Objectives: To identify the impact and predictors of the neurological complications of rheumatoid arthritis (RA).

Methodology: A series of 58 consecutive patients diagnosed as RA at Omdurman university hospital rheumatology clinic was done. Patients have another possible etiology for the neurological manifestations were excluded. A senior consultant neurologist objectively assessed the neurological complications of RA. The patients were interviewed using the Health Assessment Questionnaire (HAQ) to evaluate the functional impairment. The doctors of the rheumatology clinic examined the patients and the Clinical Disease Activity Index (CDAI) was calculated. The rheumatoid factor status and the most recent ESR value were documented. Patients' approval obtained as necessary.

Results: 60.3% have neurological signs (pyramidal signs 43.1%, extrapyramidal/cerebellar 0%, proximal weakness 8.6%, sensorimotor neuropathy 5.2%, pure sensory neuropathy 1.7%). The mean HAQ scores for patients who have neurological signs and those who don't are 1.34 and 1.40 respectively without significant difference (P = 0.778). The association between the neurological complications and disease activity, ESR value, and rheumatoid factor status is not significant (P = 0.701, 0.515, 0.299 respectively). There is strong association between the disease activity (CDAI) of RA and functional status (HAQ) of the patient (P = 0.0, R = 0.56). Moreover, 30% of variability of the functional impairment can be attributed to the variability of the disease activity (R² = 0.3).

Conclusion: The functional impairment caused by the neurological complications of RA is negligible. The RA activity is the major determinant of disease morbidity.

The disease activity, ESR value, and rheumatic factor status are poor predictors of the neurologic complications.

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215  
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Neurorehabilitation 1  
Neurocognitive interventions for breast cancer survivors: A state of the science  

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Background: Up to 90% of breast cancer survivors (BCS) report both subjective and objective neurocognitive problems following radiation/chemotherapy treatment. Many BCS indicate that these problems interfere with employment as well as quality of life. Unfortunately, only a handful of interventions have been attempted to improve neurocognitive functioning in BCS.

Objective: The purpose of this presentation was to provide a systematic overview and evaluation of the various neurocognitive interventions that have been attempted in BCS.

Method: A systematic review of the BCS and neurocognitive literature was conducted to identify published English research articles that reported on attempts to improve neurocognitive functioning in this clinical population. These studies have IRB approval.

Results: Several types of published interventions (N = 19) were identified from 2005 to 2014. Of these, they were categorized into the following groups: 1) pharmacological treatments; 2) cognitive remediation techniques; 3) compensatory techniques; 4) combination cognitive remediation + compensatory techniques; and 5) complementary/alternative medicine techniques.

These articles were examined from a methodological perspective (e.g., sample size considerations; limited follow-up time points; neurocognitive assessment) and the evidence interpreted within a neurocognitive reserve context. By and largest, most of these studies suffer from small effect sizes, while many also lack adequate control groups. Yet, most of these studies demonstrated an observable neurocognitive improvement.

Conclusion: Synergistic, combination interventions may be a way to improve the effect size of treatment since these approaches work on different mechanisms. Other novel strategies may also be considered for future research (e.g., transcranial direct current stimulation, cognitive prescriptions).

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216  
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Neurorehabilitation 1  
Neurorehabilitation in schizophrenia  

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Neurorehabilitation in patients with Schizophrenia is a set of activities capable of maximizing recovery capabilities through the development of it in their family, work and social sphere, and minimize the effects that arise from the chronicity and the natural course of this disease. This process emphasizes all the cognitivas both
individual capacities as motor through an approach to vocational, residential, recreational, social and educational support, tailored to the unique demands of the patient, in every situation and custom mode.

For this, working through concepts such as social cognition and insight, interdisciplinary way, so to set goals and accomplish them with a specific planning.

Schizophrenia is a chronic disease that occurs in 1 to 2% of the world population, and causes social marginalization and stigmatization in patients who have it. Be treated, then, since the integration of multidimensional therapeutic resources.

In this paper descriptively explains the techniques and activities developed individually for each professional and interdisciplinary area of building situational and individual patient diagnosis, potential and limitations as well as the specific treatment that may lead to improved cognitive and motor skills of the patient, and from it an improvement in their social inclusion and quality of life.

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