Is the Neural Control Different in Complex Shoulder Instability Patients?

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Introduction: The pathogenesis and etiology of complex shoulder instability remains unclear. In the absence of structural pathology, alterations in local muscle recruitment patterns may reflect a function of the behavioural context and brain activation patterns. Three different modalities were employed to examine cerebral activations, structure and the physical manifestations of shoulder movement. Functional MRI (fMRI) enables the examination of specific cortical areas involved in the production and control of motor function. MRI Diffusion Tensor Imaging (DTI), is increasingly used to quantify structural properties of the brain, using information about water diffusivity to make inferences about white matter (WM). Electromyograph or “EMG”, is used to measure muscle activation, which is the end product of that neural pathway. Both DTI and fMRI have not been used to assess conditions such as complex shoulder instability previously.

Methods: Sixteen patients with atraumatic/non structural complex shoulder instability of Polar Type III² (mean age 23y) and 16 healthy controls (mean age 24y) were studied in a Siemens Symphony 1.5 Tesla and 3 Tesla MRI scanner. For the fMRI data, acquisition subjects undertook a randomized sequence of forward shoulder flexion and abduction during fMRI. For DTI, we employed a single-shot spin-echo 50 slice, 65 gradient directions, (b value 1000 s/mm², resolution, TR 8000 ms, TE 111, interleaved slice acquisition, base resolution 1024 x 1024).

fMRI and DTI data were analyzed using Brain Voyager software with a General Linear Model (Brain Voyager, Maastricht). For the DTI data fractional anisotropy (FA) maps were calculated, then transformed into Talairach space for comparative analysis (see example in Figure x). FA reflects the degree of alignment of cellular structures within fiber tracts (i.e. WM), as well as their structural integrity.

Shoulder muscle activation patterns and amplitudes were assessed whilst subjects undertook a functional task under the FIT-HaNSA [2] protocol. The signals were recorded using a Telemyo 2400 G2 Telemetry System in conjunction with MyoResearch XP Software (Noraxon Inc, Arizona, USA). The EMG signal was differentially amplified, digitalised and band-pass filtered in accordance with international guidance.[3] Surface electrodes were utilized to record the activity of 10 muscles: trapezius, serratus anterior; pectoralis major; biceps brachii; lattissimus dorsi, teres major, infraspinatus, anterior, middle, and posterior deltoid.

The subjects completed the Western Ontario Shoulder Instability Index, the Oxford Instability Shoulder Score and a Beck’s depression inventory.

Results: The level of cortical activation of the patient group was significantly higher, both in terms of the number of regions and level of activation. Further, overall there was a difference between the centres of gravity of the activation areas, with the patient group showing more disjointed multiple centres within
the same cortical area (Figure 1). Unique to the patient group cortical activations are the premotor cortex (Brodmann 6) and somatosensory association cortex (Brodmann 7). The pattern of activation in inferior frontal gyrus (Brodmann 47) was not seen in the control group.

In the WM a difference in the FA values was found between the two groups; with a large significant difference (p=0.013) concentrated in the corpus collasum, which is the interconnection area of the premotor, supplementary motor and motor cortex (Figure 2).

Significantly greater activity (mean ± SEM) was seen in the latissimus dorsi during both phases of the movement protocol in the patient group: Phase 1 - 52.8% ± 9.1 vs 21.3% ± 6.7 (p-value 0.017); phase 2 - 52.8% ± 11.9 vs 18.9% ± 5.6 (p-value 0.044). No significant differences were identified in the other muscles of study.

In the patient group the mean Oxford Shoulder Instability Score (48 - normal) was 17.5 ; the mean Western Ontario Shoulder Instability Index (Worse 2,100) was 1164 and the mean Beck's Depression Inventory score was 12 (0-42). In the control group all the shoulder stability scores were normal, and the mean Beck’s depression score was 3.2 (0-16).

**Discussion:** There were differences in cortical activation, WM connectivity and muscle activation within the patient group. In the patient group there was greater diffused activation, and activation of different areas, such as Brodman area 47, (an area is responsible for inhibition of movement), may have a role in shoulder instability. The differences observed in the WM may equally causative of the pathophysiology of the instability. The great activity of latissimus dorsi within the patient group is the physical manifestation of the difference in neural control and confirms clinical observation in treating this patient group.

These results raise the possibility that cortical activation, WM connectivity and muscle patterning might be changed as part of the treatment of Polar Type III shoulder instability.

**Significance:** This is the first combined DTI, fMRI and EMG study of patients with Polar Type III Shoulder instability. The differences in cortical activation and WM connectivity of the patient group have previously not been reported.

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Figure 1: Tensor Visualisation of FA map of a healthy control subject
Figure 2 – Multi-Subject General Linear Model of the Patient Group

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