INTRODUCTION

Human pelvic floor musculature is made up of several skeletal muscles with a wide variety of mechanical functions. Pelvic muscles are unique in that they maintain an active contraction, even at rest [1]. This baseline activity is increased in patients with urological chronic pelvic pain disorders (UCPPS) [2,3], and may interfere with function and generate pain. UCPPS patients may have abnormal central nervous system activity [4], but potential impairment sites that might upregulate pelvic muscle activity have not been identified. To better understand the potential cortical contributions to pelvic muscle activity, we present a study to identify the neural substrates that mediate voluntary and involuntary pelvic muscle contraction.

Previous transcranial magnetic stimulation (TMS) studies have shown that the stimulation of medial regions of primary motor cortex (M1) leads to pelvic muscle contraction [5]. However, functional magnetic resonance imaging (fMRI) studies have found the predominant activation of supplementary motor areas (SMA) during voluntary pelvic muscle contractions with only sporadic reports of M1 activation [6,7]. In this study, we use TMS to show that either medial M1 or SMA activation is likely sufficient to cause an involuntary pelvic floor contraction. We additionally use fMRI to show that medial M1 and SMA are likely involved in voluntary pelvic floor contraction.

METHODS

Motor and premotor cortex was stimulated using a Magstir 100 magnetic stimulator (Magstir, Whitland, UK) with a double cone coil. Stimulation was applied at seven different locations (Figure 1) along the midline of the brain. The stimulation locations obtained from Magstir software were registered with T1-weighted structural images of brain, obtained by performing MRI of the head with 3T Siemens Trio (Siemens AG, Munich Germany). The probability of a stimulation location belonging to M1 or SMA region was obtained from the Harvard-Oxford cortical structural atlas in FSL software (FMRIB, Oxford, UK).

Figure 1: Stimulation sites represented on the T1 images of brain obtained from MRI. The orange and blue regions represent M1 and SMA respectively.

EMG response was obtained from pelvic muscles by using a rectal EMG sensor (The Prometheus Group, Denver, USA). The TMS was applied for ten times per location. An average EMG response was obtained corresponding to each location to obtain the mean motor evoked potential (MEP, Figure 2a) magnitude, as the peak-to-peak voltage, expressed in millivolts.

fMRI data was collected using a 3T Siemens Trio from nine men (mean age of 35 years). To identify brain regions involved in pelvic muscle contraction, participants voluntarily contracted pelvic muscles to the sound of a metronome during a set of fMRI data acquisition runs. Analysis of fMRI data was performed in FSL software, seeking voxels that were significantly correlated to pelvic
contraction. Statistical inferences were drawn at the voxel level with p < 0.05 corrected for multiple comparisons.

RESULTS AND DISCUSSION

Largest MEP magnitude was obtained at location 2, with a 59% probability of being in M1 and 0% in SMA (Figure 2). Stimulation at location 4, with 84% probability of being in SMA, had the highest MEP magnitude among the locations having 0% probability of being in M1. The results obtained from M1 stimulation are in agreement with the previous TMS studies showing activation of pelvic muscles on stimulation of medial region of primary motor cortex [5].

The fMRI results showed activation of both M1 and SMA regions in relation to the voluntary pelvic muscle contractions (Figure 3). The SMA activation is in agreement with the previous fMRI studies [6,7] and M1 has been found active in some fMRI [6] studies on voluntary pelvic muscle contractions.

None of the previous TMS studies have compared the SMA and M1 stimulation with respect to the pelvic muscles contraction. Our fMRI and preliminary stimulation results indicate a strong representation of pelvic floor muscles in M1 regions, along with SMA.

It has to be noted that the MEP magnitude peaks in the M1 region and then declines gradually (Figure 2b). The non-zero MEP magnitude at farther locations may be the result of the stimulation overflow. Further experiments are needed to study the effects of stimulation overflow, which can be done stimulating at multiple locations in the coronal plane as well as sagittal plane posterior to M1 cortex.

CONCLUSION

These results indicate that both medial M1 and SMA are essential regions to consider in understanding compromised control of the pelvic floor in neuromuscular disorders.

REFERENCES

3. Hetrick DC et al., Neurourol Urodyn 25(1), 46-49, 2005
7. Schrum A et al., J Urol 186, 185-190, 2011

ACKNOWLEDGEMENT

We thank Dr. Beth Fisher, Ya-Yen Lee and Allie Southam for helping with the data collection.