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Hodges P[1], Schabrun S[1], Stafford R[1]
1. *The University of Queensland*

PELVIC FLOOR MUSCLES HAVE GREATER CENTRAL FATIGUE DURING VOLUNTARY CONTRACTIONS THAN MUSCLES OF THE LIMBS

Hypothesis / aims of study

There is some suggestion from human studies that the striated pelvic floor muscles fatigue more rapidly than muscles of the limbs during voluntary efforts. This is counterintuitive considering their high proportion of slow twitch muscle fibres and the expectation that these muscles must maintain continence for sustained periods. One important consideration that has not been investigated is that the fatigue reported in humans may be due to reduced excitability of the descending pathways from the motor cortex (corticospinal pathway) which is termed "central fatigue" rather than fatigue of the muscles or other peripheral elements of the motor pathway (i.e. peripheral fatigue). Central fatigue can be demonstrated as a reduction of the ability of a person to fully activate a muscle during a maximal voluntary contraction, and can be quantified when stimulation of the corticomotor pathway evokes a greater contraction force than can be achieved voluntarily. Peripheral fatigue is quantified as a reduced force evoked by supramaximal stimulation of the peripheral nerve. This study aimed to compare central and peripheral fatigue of a pelvic floor muscle (anal sphincter) and a limb muscle (biceps brachii) during repeated voluntary activation of the target muscles. A second aim was to investigate whether the activation of the anal sphincter differed between a rested and fatigued state during a cough which includes corticospinal and other descending inputs.

Study design, materials and methods

In ten healthy subjects (males-2; females-8) elbow flexion force and surface EMG responses to transcranial magnetic stimulation (TMS) and brachial plexus stimulation were measured in the right biceps brachii. Pressure and surface EMG (measured with a custom designed anal probe) were measured for the anal sphincter muscles during TMS and sacral stimulation. Session order was randomised across subjects. Subjects performed repeated maximal voluntary contractions (MVC) of the target muscles for ten repetitions of 20 s with a brief 2 s rest between each contraction. Subjects were provided with visual feedback of force and were strongly verbally encouraged between repetitions. During the first and last sustained MVC, three TMS stimuli were delivered at 5 s intervals. Supramaxial peripheral nerve stimulation was repeated 3 times before the commencement of the trial and after the completion of the fatigue protocol to investigate the degree of fatigue of the peripheral elements. Subjects were asked to perform a "moderate effort" cough before and after the fatigue protocol and the force of the cough was measured as the peak mouth pressure recorded with a pressure transducer attached to a face mask. Central fatigue was quantified during each MVC as the amplitude of superimposed twitch force elicited by TMS expressed as a proportion of the background force. Data were compared between trials with and without fatigue using t-tests for dependent samples.

Results

Following the fatiguing protocol, there was a small decline (11%) in the the ability to activate biceps brachii voluntarily (superimposed twitich as a proportion of background EMG increased from 0.13±0..09% to 0.24±0.1%), but this decline was not significant ($t=1.04$, $p = 0.3$). The same fatigue protocol produced a significant decline in voluntary activation (44%) of the anal sphincter muscles ($t=4.2$, $P<0.001$; the superimposed twitch increased from 0.54±0.62% to 0.98±1.02% of the background EMG). Despite the reduced ability to activate the anal sphincter and biceps muscles there was no change in amplitude of the twitch evoked by peripheral nerve stimulation ($P=0.81$), indicating the absence of fatigue of the muscle.

Despite the decline in voluntary activation of the anal sphincter muscles after the fatigue protocol, when subjects performed a submaximal cough before and after the fatiguing contractions there was no reduction in the amplitude of the force of anal sphincter muscle contraction recorded with the anal probe ($P=0.21$), but the peak mouth pressure reduced by 25 (15)% ($P<0.04$). Although the force of anal sphincter muscle contraction during the cough varied between subjects and did not differ from the force generated during the final 20 s voluntary MVC effort ($P<0.75$), it is interesting to note that the force during the cough exceeded the maximum force that could be generated voluntarily in 60% of the subjects

Interpretation of results

These findings indicate that although there was no evidence of peripheral fatigue after the fatiguing protocol for either the anal sphincter or biceps brachii muscles, there was a significant decline in the potential to voluntarily activate the muscles and this was substantially greater for the anal sphincter. This observation is consistent with central fatigue of corticomotor pathway. This was contrasted with no change in anal sphincter activity during coughing, which presumably activates pathways in addition to the corticospinal pathway. The greater central fatigue of the pelvic floor muscles may have functional relevance. One interpretation is that central fatigue increases to preserve the muscle and prevent fatigue of the muscle from voluntary efforts, thus reserving the muscle for tasks when it is needed to maintain continence. One direction for future research is to identify whether central fatigue is different in people with stress urinary incontinence, which is often associated with compromised function of the pelvic floor muscles, and whether this requires consideration in rehabilitation programs where different techniques may be required to train the muscle versus the descending inputs to the muscle.

Concluding message

These data suggest that pelvic floor muscles are more rapidly fatigued than a limb muscle, and this is due to reduced voluntary activation (i.e. central fatigue) rather than fatigue of the muscle. This may have functional significance to prevent fatigue of the muscle by voluntary efforts to preserve the muscle for functional control of continence.

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