Acute neural responses to blood flow restriction strength exercise

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Introduction. A novel strength training technique that combines light-load strength training (LST) with blood flow restriction (BFR) has been shown to illicit gains in strength similar to heavy-load strength training (HST) (Takarada *et al.*, 2000). However, the neural adaptations that mediate this increase in strength following BFR are not well understood. Surface electromyography (sEMG) consistently shows motor unit firing frequency and rate coding during BFR strength exercise to be similar to that during HST, but significantly larger than the same relative intensity without BFR (Moritani *et al.*, 1992). Given that both corticospinal and spinal regions modulate neural drive, it seems plausible to suggest that BFR strength exercise may modulate the primary motor cortex (M1) and corticospinal tract (CST) to alter the pattern of motor unit recruitment. Therefore, this study examined the contribution of the M1 and CST following a single bout of BFR strength exercise. Changes in M1 and CST excitability and short-interval intracortical inhibition (SICI) can be measured using transcranial magnetic stimulation (TMS). An increase in the amplitude of the peak-to-peak motor evoked potential (MEP; a measure of corticospinal excitability) and decreases in SICI are a form of short-term neural modulation. While no study has utilized TMS to investigate changes in neural excitability and SICI, evidence for neural modulation with BFR have been obtained with temporary ischemic limb deafferenation, an established experimental model of cortical plasticity in humans (Brasil-Neto *et al.*, 1993). Of note, no exercise was completed, and TMS examined corticospinal excitability and SICI during, or following, resting ischemic conditions. The tourniquet applied across the elbow to a pressure 25-30% higher than resting systolic blood pressure rapidly increased MEP amplitude of the muscles proximal to the tourniquet (*biceps brachii* and deltoid), that persisted (>60 min) after removal of the tourniquet (Brasil-Neto *et al.*, 1993). This increase in MEP amplitude reflects changes in cortical excitability, because subcortical and spinal excitability tested with transcranial electrical stimulation, spinal electrical stimulation, and Hoffmann reflexes, did not change.

It remains unclear if BFR strength exercise can induce rapid plastic changes similar to more traditional strength exercise techniques. Therefore, this study examined whether a single bout of BFR strength exercise could stimulate changes in human corticospinal excitability and SICI, and compared these results to more traditional strength exercise methods.

Methods. Healthy males (n=5, 23±2 yr, 180.3±2.9 cm, 75±1.1 kg) completed a balanced randomized crossover study comprising 4 strength exercise trials over 4 wk. Following an initial determination of each participants 1 repetition maximum (1 RM: largest mass lifted in a single repetition; McDonagh & Davies, 1984), the 4 interventions were: HST exercise (80% 1 RM), LST exercise (20% 1 RM), and two BFR trials in combination with LST; continuous pressure application throughout the duration of the exercise bout including rest periods (BFR-C); and pressure was applied intermittently during exercise only (BFR-I). In all trials, subjects performed 4 sets of unilateral (dominant arm) elbow flexion exercise (*i.e. biceps* curl). Prior to strength exercise, TMS was applied over the contralateral M1 to elicit MEPs (normalized to the maximal muscle response [*M*max]) in the trained *biceps brachii* at 130% above active motor threshold and SICI (3 ms). *M*max, MEPs, and SICI were measured again immediately after and at 20, 40, and 60 min post-exercise.

Results. *Biceps* curl 1 RM mass lifted was 18 ± 1.72 kg. Pressure used during BFR was 96 ± 4 mmHg and 156 ± 5 mmHg for BFR-C and BFR-I, respectively. M1 excitability was significantly elevated post-exercise until 60 min compared with baseline in all trials (P<0.05). SICI was reduced at all times points following exercise (P<0.05). However, there were no differences between trials.

Discussion. The effects of BFR during strength exercise were probably induced by modifications in synaptic plasticity between neurons, and removal of local inhibition, demonstrating short-term plasticity. The findings suggest that both BFR-C and BFR-I produce similar increases in neural excitability, and reductions in SICI, when compared with more traditional strength exercise methods. Therefore, BFR strength exercise may be a suitable exercise training method for developing strength and hypertrophy in young healthy populations, and of more importance, also in clinical populations requiring rehabilitation following brain injury.

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